ARCHIVES OF OTOLOGY.

PHYSIOLOGICAL INVESTIGATIONS OF NASAL RESPIRATION.

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(*Part II. With 7 wood-cuts.*[1])

III.

DETERMINATION OF THE QUANTITY OF WATER TAKEN UP BY THE AIR DURING ITS PASSAGE THROUGH THE NASAL CAVITY.

a.—With the Balance. (See fig. 5.)

The experiments were first made in the following manner: Two U-shaped tubes were filled with pumice-stone moistened with concentrated sulphuric acid, and interposed between the left nostril and the aspirator. During one inspiration, one litre of air was drawn through the nasal cavities. The air had, therefore, first to pass through the tubes before it was allowed to enter the aspirator. The weight of U-tube II remained constant during the experiment; at times even a small quantity of acid was drawn over by the strong current of air that passed through the tube. The nasal tube and U-tube I were weighed together; the little connecting piece was either weighed separately or together with the second U-tube. Before beginning the experiment a litre of the air of the room was conducted through the apparatus, and the weight of the tubes determined immediately thereafter. The tubes were again weighed after the aspiration of a litre of the air that had passed through the nose.

[1] Continued from p. 307, vol. xvii.

At an external temperature of 17°, I found an increase in weight of the tubes after the last weight-determination of 0.0215; this increased quantity of aqueous vapor would saturate air at a temperature of 24°.[1]

In passing through the nose the air had not, therefore, been saturated to a degree corresponding with the increase in temperature; for a column of inspired air, with a temperature of 17°, is heated to 30°, in passing through the nasal cavity, as detailed above.

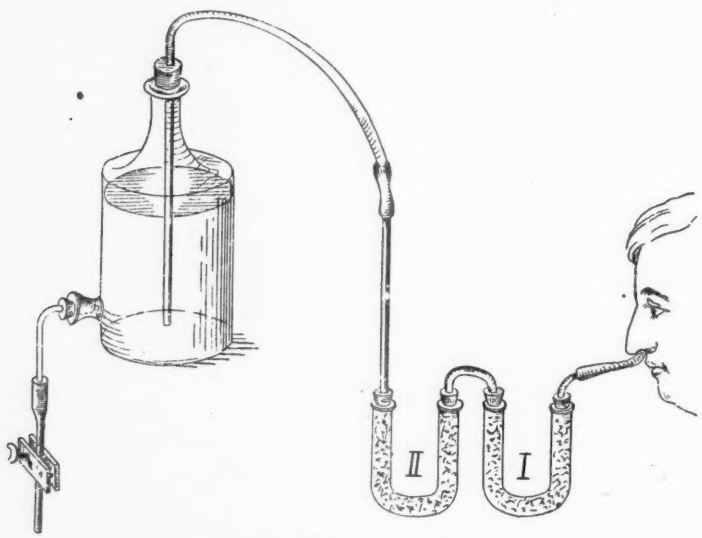

FIG. 5.

In another experiment, the external temperature was 16°; an increase of 20.8 grammes of aqueous vapor per cubic metre was found,—this weight corresponding to a saturation at a temperature of 23.4°.

In still another experiment, at an external temperature of 20°, 22.4 grammes, corresponding to saturation at a temperature of 25°. At one time a small portion of the expired air also entered the U-tube; an increase of 28.9 was recorded, corresponding to saturation at a temperature of 29°.

The last experiment demonstrated that this method of determination was not accurate. In the first place, it is

[1] Kohlrausch: "Leitfaden der prakt. Physik," 2te Auflage, Leipzig, 1872, S. 207.

difficult to obtain exactly one litre of air; besides, the velocity of the current of air is too great, especially at the beginning of the experiment, as is also the duration of each inspiration. Therefore I determined to change my method of experimenting and to employ a plan which would enable the experimenter to ignore the quantity of air aspired and the duration of the aspiration.

b.—Method of Determining the Dew-Point.

A solid which radiates heat in an atmosphere saturated with the vapor of water becomes covered with moisture as soon as its temperature falls below that of the surrounding atmosphere. If now this body be heated again, the moisture condensed on its surface disappears as soon as the temperature of the body rises above that of the surrounding medium. The temperature at which the vapor was condensed upon the body, or what is the same thing, the temperature at which the condensed moisture began evaporating from its surface, is called the *dew-point*.

Daniell's hygrometer, an instrument for determining the dew-point, consists of a glass tube terminating at each end in glass bulbs. One of the bulbs is partly filled with ether, and a thermometer inserted. A piece of cloth is wound around the other bulb, and in order to lower its temperature ether is poured over the cloth. The apparatus contains no air—only the vapor of ether. As the vapor of ether condenses in the bulb which is being continually cooled, the ether in the bulb containing the thermometer continues to evaporate; the temperature of the bulbs falls, and at a particular point the vapor of water in the surrounding air is condensed on the outer surface of the bulb containing the thermometer. The reading of the thermometer at this point gives the dew-point. The small apparatus (shown in fig. 6) has been constructed on this principle. A laryngeal mirror, 1.5 *cm* in diameter, is inserted into a small spindle-shaped glass tube; the mirror was introduced into the interior of the tube through an opening made near its middle, and then the tube was hermetically sealed; the mirror is placed obliquely to the axis

of the tube. A second tube is blown into the first, perpendicularly to the plane of the mirror, and its upper end closed by means of a cover-glass. Looking down this tube through the cover-glass, the eye of the observer will be reflected from the mirror. If moisture is condensed on the mirror-surface the image will of course be effaced. To the spindle-shaped tube a short rubber tube is attached leading to one nostril of the subject to be experimented on, while to the other end a longer rubber tube is attached leading to the mouth of the same subject. The apparatus is analogous to the one shown in fig. 3 for temperature determinations.

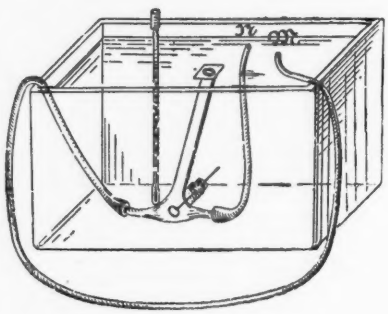

FIG. 6.

The apparatus is immersed in a water-bath, and then an inspiration taken through the tubes. The tubes very soon assume the temperature of the surrounding water, and as soon as this temperature becomes lower than the temperature corresponding to the saturation of the air, the mirror becomes covered with moisture, and the dew-point is determined. Even a small quantity of air suffices; respiration may go on normally during the experiment. The tubes were immersed in a water-bath at 26.5°; the temperature of the external air 15°.

A.—The experiment was begun at 11.15.

At 11.37,	temperature	of	bath 26.4°,	no condensation	on mirror.
" 11.40,	"	"	26.2°	"	"
" 11.42,	"	"	26.0°	"	"
" 11.45,	"	"	25.7°	"	"

At 11.52, temperature of bath 25.2° no condensation on mirror.
" 11.59, " " 24.4° " "
" 12.06, " " 24.1°, condensation on mirror.
" 12.15, " " 24.2° " "

Dew-point, therefore, lies between 24.4° and 24.2°—say, at 24.3°.

B.—External temperature of 13.5°; apparatus immersed at 4.15; temperature of water-bath 26°.

At 4.24, temperature of bath 25.4°. no condensation on mirror.
" 4.30, " " 24.5°. trace of moisture on mirror.

Apparatus dried; and again placed in the water-bath at 4.53; temperature of the water being the same as at 4.30, *i. e.*, 24.5°.

At 4.58, temperature of bath 24.4°, no condensation on mirror.
" 5.01, " " 24.1°, condensation on mirror.
" 5.06, " " 24.2° " "

Dew-point, therefore, 24.3°.

The same result was obtained with an external temperature of 12.5°.

The conclusion which we must arrive at, is that the evaporation of water from the nasal mucous membranes is independent (within limits) of the temperature of the external air.

If the air had been completely saturated while passing through the nose, the dew-point would have been 29°; for, as has been shown, an atmosphere of 23°, after passing through both nares, is heated up to 29°.

But at 29° the vapor-density of the air is . 28.63;
at 24° only . . . . . 21.59.

The air therefore has taken up only $\frac{3}{4}$ as much moisture as it would have absorbed had it been saturated for its temperature.

This method of determining the vapor-density is open to the same objections that were made to thermometric determinations. All we can assume, in view of the results that have been obtained, is, that the air in passing through the nose absorbs moisture in some direct proportion to the

resulting rise in temperature. If we determine the thermometric action in the nasal cavity of one side according to the formula

$$E = \frac{5}{9}(37 - t)$$

we may calculate the amount of water absorbed according to the same formula; we can also assume that $\frac{1}{6}$ less of the vapor of water is absorbed by the air in passing through one nasal cavity than there would be in passing through both. Instead of the fraction $\frac{75}{100}$, we obtain therefore for the vapor-density of the inspired air, after passing through the nasal cavity of one side, the smaller fraction

$$\frac{63}{100} \left(\frac{5}{6} \text{ of } \frac{75}{100}\right).$$

Therefore the air, at an average temperature, would pass into the pharyngeal cavity through the nose only $\frac{2}{3}$ saturated.

IV.

RETENTION OF DUST-LIKE IMPURITIES IN THE INSPIRED AIR BY THE NOSE.

How small particles of dust contained in the air are retained in the nose has, to my knowledge, not yet been examined. Is it due to the viscid condition of the mucous membranes, or is it to be attributed to the weight, or the hygroscopic condition, or perhaps the solubility of the solid particles? Is it, moreover, true that all particles of dust, provided they are coarse enough, are retained in the nose? This view is held by Aschenbrandt.

It seemed desirable to investigate this interesting question, to ascertain whether or not particles of dust are retained in the nose during the act of inspiration, and, if retained, to what extent.

For these experiments I used the following substances: Wheat flour, potato flour, powdered cloves, lycopodium, chloride of ammonium, boracic acid, kino, shellac, magnesia, gypsum, chalk, charcoal, anthracite coal, burnt umber, sienna, sap-earth, etc.

First I examined specimens of these powders under the microscope (with a linear enlargement of 138), and then

took 1 *cc* or 10 *cc* of each powder, weighed it, and observed also how each substance reacted on warm and cold water.

The experiments were conducted in the following manner (see fig. 7):

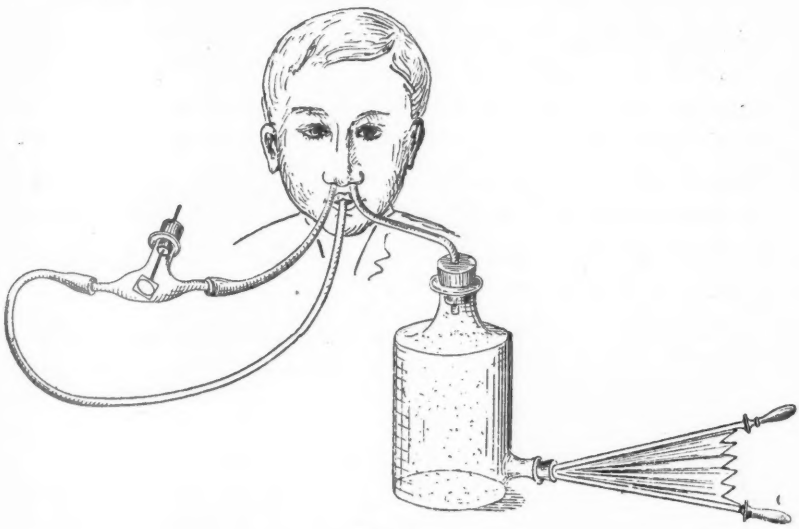

FIG. 7.

The powder was placed into a large bottle provided with a rubber stopper, and was stirred up by means of bellows. Through the stopper a glass tube was inserted, connecting with a rubber tube, which terminated in a glass nose-piece for the right [1] nostril. The apparatus which had been used to determine the dew-point was connected through a similar arrangement with the left nostril. The mirror was inserted less obliquely than in the apparatus before described, and was covered by a cover-glass whose upper surface was moistened with glycerin; the distal end of the apparatus passed, as in the other experiment, into the mouth of the subject.

The inspired air first passed through the bottle, then through the nose from right to left,[2] then past the cover-glass in the apparatus, and finally into the mouth and lungs. Each experiment was limited to one inspiration, and at its

[1] In the drawing the nose-piece is inserted into left nostril.
[2] In the figure from left to right.

conclusion the cover-glass, as well as the mucus blown from the nose, was examined under the microscope.

1. *Wheat Flour.*

The right nasal cavity, *i. e.*, the cavity through which the air passed first, is found to contain starch, the presence of which is determined by the iodine test; the secretion from the pharynx gives the same reaction. The left nasal cavity contains but very few granules; on the cover-glass, *no trace of amylum visible.* This experiment was repeated several times, and with uniform results. One *cc* of the flour weighs 0.76 gramme. Starch granules are very hygroscopic; swell up in water.

2. *Potato Flour.*

Potato-starch granules are four to eight times as large as those of wheat; their size, however, varies considerably. They swell in water somewhat more than do wheat granules. One *cc* weighs 0.91 gramme. This is one of the heaviest of all the powders employed. The secretion from the right nares was found to be very rich in amylum cells; the pharyngeal secretion contained rather small granules. No amylum corpuscles in the left nasal cavity; none on the cover-glass.

3. *Lycopodium.*

Round, dark-yellow granules of uneven surface, of nearly uniform size (four times the size of wheat granules). One *cc* weighs 0.42 gramme; lycopodium is absolutely non-hygroscopic. The right nasal cavity, as well as the pharynx, contained a few granules; left contained many. The cover-glass also showed several grains of dark and light colors.

4. *Powdered Cloves.*

Very dark granules, fragmentary, and in groups; the single cells are not always recognizable as such; powder not very hygroscopic; one *cc* weighs 0.60 gramme. The strong scent of cloves appreciable only in the right nasal cavity, and a large quantity of powder found on this side; only traces, however, in the pharynx and the left nasal cavity. On the cover-glass were found several small grains.

5. *Dragon's Blood.*

A resin, obtained from Dracæna draco, of reddish-brown color; is not changed by glycerin; under the microscope the resin appears to consist in part of regular small granules, and in part of large amorphous masses; one *cc* weighs 0.46 gramme; it is not hygroscopic; floats in water, and is insoluble. In the secretion from the right nares were found large quantities of the granules, but only of the smallest size; the same in the left; the cover-glass retained large quantities of the resin, but only smaller granules.

6. *Shellac.*

Light, amber-colored, coarse, amorphous granules, with sharp edges, as large on the average as the larger potato granules; one *cc* weighs 0.69 gramme; not hygroscopic; insoluble; floats partly on water. In the right nasal cavity many large and small particles were retained; on the cover-glass were found several small and large granules about the size of the potato-starch granules.

7. *Kino.*

Yellowish-brown, amorphous masses; otherwise very similar to the resin; soluble in glycerin; soluble in water; when inhaled gives rise to a peculiar odor, and a fæcal taste in the mouth; one *cc* weighs 0.68 gramme. Upon inspiration many granules were found in the right side of the nose, though not of the very largest size; in the left nasal cavity, several smaller ones; on the cover-glass, many of the smaller granules.

8. *Magnesic Carbonate.*

Each of the crystals is about three times as long as the large potato granules. One *cc* weighs 0.14 gramme. The powder sinks rapidly in water. Experiment produces a sensation as if the nose and pharynx had been filled with dust. A large quantity of the light powder is retained in the right nasal cavity. Nevertheless, we find many very small granules on the cover-glass.

9. *Gypsum.*

This powder is composed of rhombic tablets, and also of small particles of irregular shape. One *cc* weighs 0.93 gramme; very hygroscopic; produces the sensation of dust already described. In the mucus from the right side, a few large particles and much detritus are found; on the left, only small particles; the same on the cover-glass.

10. *Chalk.*

Settles rapidly in water; moderately hygroscopic. One *cc* weighs 0.70 gramme. On the cover-glass but few small particles are visible under the microscope; but on the addition of HCl a large number of separate bubbles appear.

11. *Burnt Umber.*

Pyrosiderite, containing clay, probably adulterated with peat. Under the microscope small dots and amorphous masses of a dark-brownish or yellowish hue are visible; very hygroscopic; one *cc* weighs 0.61 gramme; insoluble in cold water. The inhalation of this powder produces a disagreeable irritation in the throat and excites coughing. The mucus from the right as well as from the left nares is very much colored; on the cover-glass many of the smallest particles.

12. *Sienna.*

Round yellow granules, about the size of wheat starch; is composed of clay, ferric hydrate, and manganese; one *cc* weighs 0.84 gramme; very hygroscopic. The inhalation produces a sensation of dust in the throat. On the right side, but few heaps and many single particles; the same on the left; on the cover-glass, numerous of the smallest granules.

13. *Sap-Earth.*

This powder is composed of silica, iron, and magnesia; under the microscope, amorphous masses of olive-green color. One *cc* weighs 0.60 gramme. Very hygroscopic; gives a muddy filtrate. Upon respiration, an earthy taste on the tongue. The right side contains very many particles; somewhat less on the left; only a few of the small particles on the cover-glass.

14. *Charcoal.*

Appears under the microscope as small, irregular, amorphous granules; one *cc* weighs 0.51 gramme. The sensation of dust is produced in the nose and throat. After inspiration, several coarser granules on the right side; smaller on the left; on the cover-glass an innumerable quantity of the smallest-sized pieces, as well as a few larger particles.

15. *Anthracite Coal.*

Finely powdered anthracite coal was used; the granules are somewhat sharper on the edges than those of charcoal. One *cc* weighs 0.74 gramme. Somewhat hygroscopic; settles very rapidly in water. On inspiration, a sensation of dust in nose and throat. In the right and left nasal cavities, many small granules and also several larger ones.

16. *Salicylic Acid.*

Produces the well-known irritation in the throat when inhaled through the nose. Passes through the nose easily in spite of the fact that single crystals are rather large.

17. *Boracic Acid.*

Acts similarly. The same sensation in the nose; irritation in the throat not quite as great as with salicylic acid.

18. *Chloride of Ammonium* (*Vapor*).

May be readily driven through the nose either from the anterior or posterior nares; was generated with the apparatus used by Dr. Thiry in the Aural Policlinik. The apparatus consisted of three bottles connected together by short glass and rubber tubes. One bottle contained HCl; the other, ammonia; and the third, water.

Air is forced through the bottle containing HCl by means of a pair of rubber bulbs. The HCl gas is thus forced into the bottle containing ammonia; the spray of chloride of ammonium thus formed passes through the water contained in the third bottle, and is there freed from the excess of acid. From the last bottle it passes out in dense vapors.

If the vapor be pumped into one nasal cavity, it passes

out of the mouth a little less dense than when it entered; and in the same way it can be forced out of the nares when passed through the mouth. The same occurs with tobacco-smoke; a certain quantity is always retained by the nose; even in the buccal cavity the smoke precipitates in considerable quantities.

We see, therefore, *that of all the powders examined, the greater part was retained in the nose, but that, nevertheless, a certain quantity of all of them passed out*, amylum being the only exception in that it was wholly retained in the nose.

But if dust particles can pass the nares, we can assume with absolute certainty that they also enter the respiratory organs. During ordinary respiration, therefore, dust will pass through the larynx into the lungs, although a small fractional part will also be retained by the mucous membrane of the pharynx. Although the velocity of the current of air in passing from the narrow nasal passages into the wider pharyngeal space is diminished, it is improbable that all the particles can come in contact with the walls of the pharynx.

The powders used in the experiment showed considerable differences in their physical properties; the weight varied from 0.14 gramme (magnesia) to 0.93 gramme (gypsum) per *cc.*

It is quite evident that the fact of starch being wholly retained was not due to its specific gravity; nor does the condition of the surface of the particles afford us any explanation therefor. Amorphous, crystalline, pointed, rough, or smooth particles, all passed through the nose; starch was even the smoothest of all.

Nor can we explain this phenomenon on the ground of the differences in the size of the particles, although it is true, in a general way, that the smallest granules pass through the nose most readily. Nor does the hygroscopic condition of the substances used afford us an explanation, although it is true that shellac, dragon's-blood, lycopodium, etc., substances which are least hygroscopic, passed through the nose in great quantities. Still charcoal, which is very hygroscopic, also passed through very readily.

Nor is it true that the more hygroscopic bodies are retained by the nose when they have once come in contact with its mucous membrane.

A simple experiment proves the contrary: powdered amylum and dragon's-blood were well mixed, and some of the powder was strewed over a piece of moistened paper; the back of the paper was then flapped with the finger several times, and a part of the powder naturally shaken off. The paper was then examined under the microscope. Both granules of dragon's-blood and amylum granules were visible. If lycopodium be used instead of dragon's-blood, even more lycopodium than starch granules are retained.

The reason that starch is retained more than other substances is probably its *relatively high specific gravity, together with its enormous capacity for the absorption of water.* Its weight is probably so much increased by the water which it absorbs, that all or very nearly all the granules precipitate during their passage through the nose.

On the whole, therefore, we may assume it to be true in a general way, that the amount retained will depend somewhat on the weight, the hygroscopic property, and the size of the particles.

We come to the conclusion **that small solid particles are retained by the nose to a great part, but that the nose is not capable of freeing the inspired air entirely from impurities, not even with the aid of the naso-pharyngeal cavity.**

V.

THE EFFECT OF NASAL RESPIRATION ON THE BUCCAL CAVITY.

The preceding remarks have clearly shown the advantages of nasal respiration. A current of air inhaled through the mouth causes too rapid a loss of moisture in the buccal and pharyngeal cavities, since their mucous membranes are not provided with the natural apparatus for replacing the water lost by evaporation.

The cavities also lose heat too rapidly, and their mucous

membranes have not the mechanism provided to replace this loss. The teeth must also suffer in this unnatural form of respiration, and in this way even further disturbances, such as impairment of smell, taste, etc., are possible.

I believe also that nasal respiration is not without influence on the interchange of gases in the lungs. We need not dwell any further upon the fact that the larynx, the bronchi, and the lungs are provided with no other filter against solid impurities in the air than the nasal cavities.

I propose here to discuss the effects of nasal respiration upon the buccal cavities. Mezger [1] has called attention to the fact that, in breathing through the mouth, a special muscular effort is required, though the mouth can be kept closed for hours without any such expenditure of muscular energy. During nasal respiration, the tongue, by pressing tightly with its upper surface upon the vault of the palate, shuts off the communication between the buccal and pharyngeal cavities. The margins of the tongue and a part of its lower surface meanwhile rest on the lower jaw. The weight of the tongue and the lower jaw are supported by the pressure of the external air alone, during nasal respiration, no muscular effort being required for this purpose.

Donders [2] agrees with this view, and adds, that if we measure the pressure of the air in the mouth manometrically during nasal respiration, we find no fluctuations recorded as long as the mouth-piece remains in the buccal cavity; but if the mouth-piece, which had been attached to the manometer by a long rubber tube, is placed away back into the pharynx, the fluctuations ordinarily obtained on connecting the manometer with the nose, for instance, are immediately recorded. This experiment affords another proof that the buccal cavity is shut off from the pharynx during nasal respiration. According to Donders, the contracted soft palate acts as an obturator between the two cavities. According to the same author, the mouth is kept in position

[1] Über den Luftdruck als mechanisches Mittel zur Fixation des Unterkiefers gegen den Oberkiefer im ruhendem Zustande. Von Dr. Joh. Mezger, Amsterdam. Pflüger's *Archiv f. d. gesammte Physiologie*, Bd. x., Bonn, 1875, S. 89.

[2] "Über den Mechanismus des Saugens," von F. C. Donders, *l. c.*, 91.

during sleep, for instance, chiefly by the action of the hyoglossus muscle.

Henke[1] expressed a similar view years ago. During ordinary respiration, according to this author, the soft palate rests on the back and base of the tongue, the elongated uvula in the groove on the posterior surface of the epiglottis, while the lingual surface of the latter is in apposition with the base of the tongue, so that a direct and open passage is formed from the posterior nares to the larynx. "The current of air during ordinary respiration, therefore, does not come in contact with the buccal cavity. According to Henke, the anterior portion of the tongue is held in position by muscular action.

The following experiment proves clearly that during nasal respiration, the buccal cavity is hermetically shut off from the pharyngeal. The mouth is filled with tobacco-smoke to such an extent that the cheeks are puffed out; if the cheeks are now compressed, the lips being closed, no smoke enters the nose or the larynx; the lips may even be tightly pressed against the incisors, and the cheeks drawn in, as in sucking, and still no smoke passes beyond the buccal cavity. In order to compensate for the diminution of space in the anterior portion of the buccal cavity, the back of the tongue probably separates from the hard and the soft palate. Posteriorly the buccal cavity, during this time, is probably separated from the pharynx by the border of the palatal arch and the uvula alone; certainly not by more than a very small portion of the soft palate.

Although I have not been successful in my attempts at repeating the experiment of Donders, in measuring the pressure in the buccal cavity throughout its whole extent, I have partially succeeded in determining the pressure when the mouthpiece attached to the manometer was placed close to the hard palate; not a trace of change in the pressure was recorded. Of course, I took care that the lumen of the rubber tubing

[1] Ph. J. W. Henke: "Beiträge zur Anatomie des Menschen mit Beziehung auf Bewegung." Leipzig und Heidelberg, 1872, Erstes Heft, Anhang, "Versuche in Bildern zur Topographie der Bewegungen," Tafel ix., Bewegung des Schlundkopfes, Fig. 3; Stellung beim Athmen. S. 42.

was not obliterated by pressing it too tightly with the teeth. If the mouth is then opened, or the manometer connected with the nasal cavity, the changes in pressure during respiration become at once apparent. In those cases in which the epiglottis has a horizontal position—as is sometimes found during a laryngoscopic examination,—it is impossible that the uvula should fit into the saddle-like groove on its posterior surface, as Henke believes. I cannot agree with this view of Henke's, for I cannot imagine that in ordinary respiration the epiglottis should be in the position which we find it in during a laryngoscopic examination. Such positions are, in my estimation, artificial. But as the position of the epiglottis does not play a very important rôle in the physiology of the buccal cavity, I do not deem it necessary to object entirely to Henke's view as Störk[1] has done. The position which Henke assigns to the anterior border of the tongue is also, in my opinion, not quite correct. During nasal respiration, the tip of the tongue generally rests between the teeth in apposition with the mucous membrane of the lips or the cheeks. This can be proved by careful self-observation. At times, but by no means constantly, are the teeth kept closed. Nor do I believe that the mouth is closed at the beginning of nasal respiration by the action of the lingual muscles. When the mouth is closed, the palate and the tongue are still separated by a certain quantity of air. This residual air communicates through the nose with the external atmosphere, and is therefore of the same pressure. This air is aspired at the first inspiration that follows the closing of the mouth. As the pressure in the buccal cavity is now zero, or at any rate has become less than normal, the positive pressure on the pharyngo-nasal surface of the soft palate brings the soft palate in apposition with the posterior surface of the tongue. In the meanwhile, the anterior portion of the tongue is pressed upon to the upper maxillary and hard palate by the positive pressure of the air under the chin.

[1] C. Störk : Klinik der Krankheiten des Kehlkopfes. Stuttgart, 1880. Erklärung zu Tafel iii., in front of page 185.

Nasal respiration thus saves an amount of work which would otherwise require the expenditure of muscular energy.

The pernicious effects of mouth-breathing may be seen in the children who suffer from adenoid vegetations. The tired, sleepy, and usually stupid expression of countenance of such children furnishes an apt illustration of its bad effects.

VI.

THE RESPIRATORY REFLEXES EXCITED BY THE NASAL MUCOUS MEMBRANE.

In 1870 Kratschmer,[1] of Vienna, discovered that when a volatile fluid—chloroform, ether, alcohol, ammonia; acetic, hydrochloric, and nitric acids, etc., or tobacco-smoke—is placed in front of a rabbit, the animal immediately makes a strong expiratory movement and closes the nostrils. The same thing happens if the outer surface of the nose is irritated mechanically by tickling, pinching, or cooling, as with a piece of snow. This reflex-expiration occurs even if the stimulus be applied at the beginning of an inspiration. In addition to this expiratory movement and the closure of the nostrils, respiration ceases, the vocal cords are approximated, and the glottis closes until the beginning of the following inspiration. At the same time the heart ceases to beat, and its function is only gradually restored, so that it does not attain its normal rate until some time after respiration is again normal. Respiration sometimes was observed to cease for one or two minutes, complete arrest of respiration lasted as long as fifty-three seconds, the average arrest being twenty seconds. Furthermore, the blood-pressure was considerably increased for a time after the application of the stimulus.

Kratschmer also showed that irritation of the larynx or of the trachea did not give rise to these reflex phenomena, but that the results were only obtained upon irritation of the mucous membrane of the nose.

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[1] F. Kratschmer: "Über Reflexe von der Nasenschleimhaut auf Athmung und Kreislauf." Sitzungsber. d. Akademie der Wissenschaften, math.-naturw. Classe, Bd. lxii., 2. Abth., Wien, 1870, S., 147 *ff.*

If the vagi are cut, there is no effect produced on the heart. Respiration and the blood-pressure are affected, however, in the manner described. If the trigeminus be cut, however, the reflexes before produced do not occur; the course therefore must be through this nerve. The olfactory nerve has no influence. In order to produce a closure of the glottis in rabbits, it is only necessary to introduce a probe in the nose; after cutting the vagi, this reflex is prevented.

Kratschmer considers this reflex to be a natural safeguard against the aspiration of substances that might irritate the respiratory organs. Respiration ceases in order to prevent the aspiration of these, and the strong expiration that immediately follows expels what may already have been inspired.

According to Kratschmer, therefore, "there must be a peculiar reflex connection between the nasal mucous membrane on the one hand and the respiratory and circulatory organs on the other." The sensory fibres of this connection lie in the trigeminus; the motor, in the nerves which supply the muscles engaged in respiration; other motor fibres for the heart, in the vagus. The reflex occurs whenever that portion of the nasal mucous membrane is irritated, whose nerve supply is derived from the trigeminus. It effects an expiratory tetanus with a simultaneous closure of the glottis, slowing of the heart-rate, and increase of blood-pressure in the arteries.

These important observations have been confirmed in a recent publication by Sandmann.[1] Sandmann, however, found that stimuli of less intensity do not always necessarily produce an expiratory tetanus, but simply cause an arrest of respiration in its particular phase at the moment of irritation.

Sneezing is the reflex that results on tickling the mucous membrane of the nose. The reflex is as follows: a deep inspiration, upon which the naso-pharyngeal space is shut

[1] G. Sandmann: "Über Athemreflexe von der Nasenschleimhaut." Verhandlungen der physiolog. Gesellschaft zu Berlin, Jahrgang 1886–87, No. 18, vom 15ten August, 1887.

off from the nose by the soft palate, the buccal cavity by the tongue; a strong expiration follows, during which the pharynx is again opened, while the air under strong pressure escapes through the nose.

The nasal cough cannot be produced in rabbits and cats. In these animals the region of irritability is confined chiefly to the anterior portion of the inferior turbinated bones and the parts of the septum adjoining them. In the human being, the sensitive zone is confined to the anterior portion of the inferior and middle turbinated, the portions of the septum adjoining, and corresponding portions of the posterior turbinated. The anterior zone is supplied by the ethmoidal nerve, a branch of the naso-ciliary—of the first branch; the posterior by the pterygo-palatine nerve—a branch of the second.

The reflexes that follow irritation of these parts are, depending upon the nature or the intensity of the stimulus: slowing of respiration, arrest in some phase of respiration, arrest in expiration.

On attempting to study these observations on the human subject, we find that the reflex produced by tickling is exactly the same as the one found by Kratschmer and Sandmann in animals. Common observation shows us that strong irritations produce inhibitory reflexes in the human subject also. Inhalation of pungent vapors, of dense smoke, a cold and sudden draught, dense dust, takes one's breath away, *i. e.*, arrests respiration. The same phenomenon is also observed during mechanical irritation, especially during operations that are performed in the nose —galvano-caustic operations, for instance. If the individual is very sensitive, the mere introduction of the speculum produces the reflex. I have very often observed the occurrence of a quick expiration, apparently produced by reflex action, upon the application of the galvano-cautery. Immediately upon cauterization, the smoke produced is expelled by a sudden expiration; if the parts are first anæsthetized with cocaine, the reflex does not occur.

Experimentally, I have succeeded in obtaining this inhibitory reflex only during inspiration (and during any

phase thereof), but not during expiration; for very strong irritations would be required to produce this reflex during expiration, too strong for experiments on human subjects.

A cylindrical tube, 3 *cm* in diameter and 8 *cm* in height, is curved into the shape of a segment of a circle of very large radius. A short tube, 4 *mm* in diameter, inserted into the middle of the convex side, serves to connect the cylinder with a Marey's tambour-à-levier. The ends of the

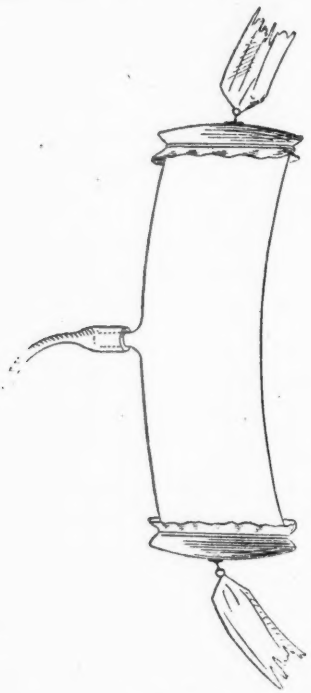


FIG. 8.

bent tube are hermetically sealed with fine rubber-membranes provided with metallic rings for the attachment of elastic bands. This apparatus was fixed to the thorax, on a level with the lower end of the sternum, by means of the elastic bands. The index of the tambour records the respiratory movements of the thorax upon a drum, which is set in motion by clock-mechanism, the movements of the thorax being communicated to the index through the expansion and contraction of the elastic bands and the air

contained in the cylindrical tube. At every inspiration the elastic belt pulls the rubber membranes outward, the area of the cylinder is enlarged, the air accordingly rarefied, and the index falls. At every expiration the opposite takes place, and the lever-index is raised (fig. 8).

If, now, during nasal respiration, a pungent vapor like ammonia be inhaled, the index records a horizontal line: respiration has come to a standstill. This cessation of respiration may occur at the beginning, during, or at the end of an inspiration, depending on the phase of the inspiration at which the stimulus was applied in the nasal cavity.

If the nostrils are closed, and the vapor is inhaled through the mouth, this inhibitory reflex does not occur.

If ammonia is sprayed into the nose during expiration, the arrest of respiration is not recorded before the ensuing inspiration begins. Immediately after the application, I felt a burning sensation in the floor of the nasal cavity; but the expiratory current of air probably prevented the gases from coming into too close contact with the sensitive zones. This zone was, however, irritated upon the ensuing inspiration.

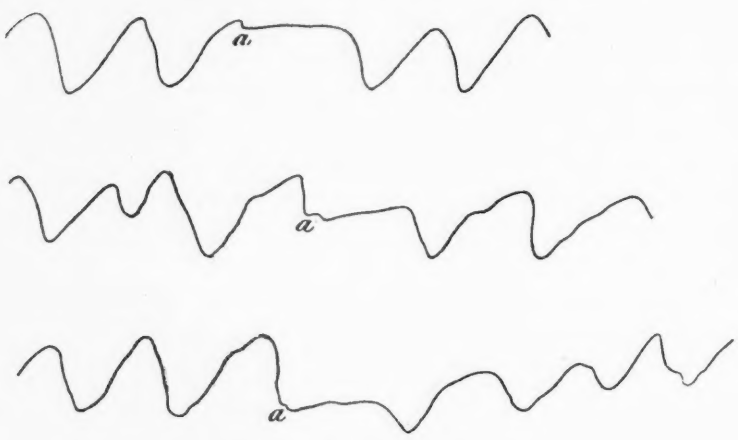
Tobacco-smoke in greater quantities has a similar effect; as also has acetic acid, chloroform, etc. The duration of the inhibitory action is, within limits, proportional to the quantity of the vapor inhaled, *i. e.*, proportional to the intensity of the stimulus.

Attempts to influence respiration by means of faradic stimulation of the nasal mucous membrane yielded no positive results. A wad of moistened tissue-paper, around which the ends of a conductor had been wound, was shoved into one nostril between the septum and the inferior turbinated bone, while the other electrode was held in the hand. Although the current was increased to such an extent as to become positively painful, the curve recorded by the tambour-lever was not altered in the least.

The respiratory standstill was invariably followed by an inspiration, even though the arrest took place at the end of the previous inspiration.

The curves shown in Figs. 9, 10, and 11 illustrate the results obtained :

Stimulus, ammonia. Inhibitory reflex begins at *a*.



FIGS. 9, 10, 11.

As already stated, spasm of the glottis, in all probability, generally accompanies the pause in respiration,—to judge from the muffled tone of the voice. Whether this spasm is due to the fact that expiration becomes more difficult, or whether the arrest of the inspiration may not be due in part to the ensuing spasm, we must leave to further investigation. I have, however, satisfied myself by direct observation, that during oral inspiration a large quantity of ammonia may be inhaled, without any effect on the glottis whatsoever. In oral inhalation, moreover, respiration does not cease ; the record shows simply a slight retardation and a diminished intensity of the respiratory movement. This retardation however may be due to some psychical influence. The experiment proves positively that a spasm of the glottis is dependent solely on the irritation of the nasal mucous membrane.

If the nasal mucous membrane be but slightly irritated, as, for instance, with the probe, and the larynx examined at the same instant, we find that there is no approximation of the vocal cords toward the middle line. It is impossible to study the effects produced in the larynx when very strong

stimuli are applied, because a reflex contraction of the pharyngeal muscles accompanies the (probably) ensuing spasm of the glottis, and precludes the possibility of obtaining a laryngeal image.

This much, however, is certain: *that in the human subject a strong mechanical, thermic, or chemical stimulus applied to the nasal mucous membrane during inspiration produces cessation of respiration in whatever position the thorax may happen to be at the moment the stimulus is applied.*

I have not been able to determine whether or not the influence exercised on the action of the heart by stimulation of the nasal mucous membrane is the same in the human subject as in the animals upon which the experiments have been made. I have observed, in some cases, a retardation of the pulse after the application of the galvano-cautery; in others, again, this retardation did not ensue. The late Prof. Hack also called attention to this phenomenon. We cannot, however, assert positively that in these cases the heart has been acted on merely by painful irritation of the trigeminus, as we know that irritation of almost any sensory nerve produces a similar effect on the heart's action.[1]

My experiments, made especially with a view to determining these reflex effects on the heart-action, were without positive results. Stimuli which brought respiration to a standstill had no marked effect on the recorded pulse-curves; my experiments in this direction, however, were brought to an untimely end by the vertigo, irregular heart-action, etc., which ensued.

I believe the experiments on the physiology of nasal respiration to warrant the following conclusions:

1. *The temperature of the inspired air is considerably raised while passing through the nasal cavities.*

2. *The thermogenic action of the nasal mucous membrane is greater when the temperature of the external air is lower.*

[1] See Francois-Franck: Effets des excitations des nerfs sensibles sur le cœur, etc. Physiologie Expérimentale, Travaux du Laboratoire de Marey, II. Année, 1876; Paris, 1876, S. 221 *ff.*; also Recherches sur le méchanism des accidents cardiaques causés par les impressions douloureuses (*Gaz. hébdom*, 1876; cit. *Schmia's Jahrb.*, 1878, Bd., clxxvii).

3. *The thermogenic effect of the nasal mucous membrane is constant, depending on the relation of the external temperature to the temperature of the body.*

4. *Under normal conditions this relation is expressed by the formula* $E = \frac{5}{9}(37 - t)$.

5. *The temperature of the expired air is* 1.5°–2° *C. higher after nasal respiration.*

6. *The thermogenic action of the buccal cavity is slight as compared to that of the nasal.*

7. *The heat given up by the nasal mucous membrane at each ordinary inspiration, and at a moderate external temperature, is equivalent to* 6 *gramme-calorics.*

8. *Inspired air passes out of the nose about* $\frac{2}{3}$ *saturated.*

9. *The nasal mucous membrane retains the greater part of the solid particles which may be contained in the inspired air.*

10. *It does not, however, act as a perfect filter for dust.*

11. *The amount of dust retained depends principally upon the normal viscidity of the nasal mucosa; also on the weight, size, and hygroscopic condition of the solid particles inhaled.*

12. *During nasal respiration, the tongue is pressed against the palate, and the mouth closed anteriorily and posteriorily, solely by the pressure of the external air.*

13. *Every considerable mechanical, chemical, or thermic stimulus, acting upon the nasal mucous membrane through the inspiratory air-current, induces an immediate cessation of the respiratory act.*

14. *This respiratory standstill is immediately succeeded by an inspiration.*

15. *It is probable that weak stimuli only retard inspiration; and it is possible that very strong stimuli induce an immediate expiratory movement.*

REPORT OF CASES OF DISEASE OF THE MASTOID PROCESS, WITH REMARKS.[1]

By HENRY FERRER, M.D., San Francisco, Cal.

Case 11.—**Chronic Otorrhœa, left side, with Caries of Mastoid Extending to Occipital Bone; Chiselling of Mastoid; Phthisis Pulmonum; Death.**

Mr. N——, about 35 yeara old. I saw him first the 3d of May, 1886, in consultation with Dr. Barkan, who had been treating him a couple of weeks for otorrhœa of left side. He complained of severe pain in the ear, forehead, and occipital region, the discharge was very profuse, there was slight swelling of walls of meatus, no swelling of mastoid, but sensitiveness upon pressure. He kept his bed, had fever and night-sweats, cough and mucopurulent expectoration. The doctor kindly advised him to go to the German Hospital and place himself under my care. The following day he was admitted, and treated with inflations of middle ear either with Politzer's method or catheter, syringing of meatus a couple of times daily with a 1 % solution of carbolic acid; the temporal and mastoid regions were shaved, and a strong tincture of iodine and an ice-bag applied. This treatment was continued until the 17th; in the meantime we had found that both lungs were extensively infiltrated at apices, and tubercle bacilli found in sputum. Several attempts to find the bacillus in the pus from meatus failed. The night-sweats were profuse, for which we gave him large doses of morphia. The ice relieved him, but now there was wellmarked swelling of mastoid; the pain had become excruciating and we decided to operate.

Operation: Dr. Barkan and Dr. J. Morse assisting. Incision of external integuments as usual, and about 5 to 6 *ctm* long. Peri-

[1] Continued from p. 326, vol. xvii.

osteum was attached to the bone, the surface of which was slightly discolored. Integuments about 0.1 thick, infiltrated; moderate hemorrhage. *Spina supra meatum* and emissary vessels not well defined. I began to chisel as usual with the largest chisel, and at the depth of 2 to 3 *mm* pus was found; after enlarging the external opening the scoop was mostly used, the trabeculæ and even the external plate felt spongy. The quantity of pus found was much smaller than expected. I penetrated over 2½ *ctm*, and in trying forced injections no communication had been obtained. The same night patient had considerable pain and more or less discomfort until June 5th. The discharge from meatus had decreased; the granulating process was so rapid and the granulations so pale that on 5th of June I decided to scoop them out again and to introduce a nail. The reason that no nail had been used before was the continual complaint of pain, which was only relieved by the constant use of ice and large doses of McMunn's elixir of opium. I need not state that he had been attended once and even twice daily, and that severe cauterization of the granulations had also been resorted to.

In this second operation only the scoop was used, and an effort made to obtain communication; the depth reached was 3 *ctm*. In very few days granulations began to shoot up, and a conical lead nail was applied. Patient still complained of pain, especially in left occipital region. This part became very sensitive to pressure, and an abscess formed about 5 *ctm* backwards and 3 below the original opening in the mastoid. On the 26th of July he was placed again under ether, the abscess opened, discharging a tablespoonful of pus; the original opening in mastoid was scooped out again. We then discovered that most of the mastoid appendix had been removed; the softening of the bone extended into the occiput and materially affected even its surface. The bone was spongy and filled with granulations. A thick drain-tube was carried through from the opening in the occiput to that in the mastoid and was thoroughly cleansed. Communication with middle ear had been established and the discharge decreased very much. During the month of August he recovered materially, was able to be up most of the time and to walk in the hospital grounds. He left on the 17th. Afterwards he was attended to in my office as an out-door patient until September 18th. The drain was removed; the canal connecting occipital abscess and mastoid had closed perfectly; there was very little discharge from

the meatus and mastoid. Here a smooth canal existed of the exact shape of the nail. He had learned how to attend to his own case, using a Davidson syringe with a short point to cleanse the meatus as well as the fistula in mastoid. His general health, though somewhat improved since the last operation, was much worse than when I first saw him. He was fortunately able to take, besides three good meals daily, three and four tumblers of an emulsion of cod-liver oil and malt with whiskey; he had even reduced and often done without morphine at night. His cough had somewhat decreased, the expectoration was not profuse, and in this condition he left, somewhat hopeful, for the mountains.

February 5th I received a letter from Dr. Rooney, in Auburn, Cal., stating that he had been attending to Mr. N——, who was taken with paralysis of the tongue and muscles of deglutition, produced by post-pharyngeal induration, which he incised, liberating a large quantity of pus. In syringing the abscess with carbolized water, communication existed with the external meatus. The injections were used daily; patient could then speak again and take food, only complaining of intense pain. He died the first of March.

The question arises whether it was advisable to perform the primary operation, and, if this was the case, whether or not there was indication enough for the second and third operation. There is no doubt at all that we had to deal with a case of tubercular affection of the temperal bone, beginning probably with an acute otitis media purulenta and gradually affecting the neighboring portions of the mastoid and occipital bones. All my attempts, with different methods of staining, to find tubercle bacillus in the scooped tissues after each operation failed. The primary operation was performed to give the incarcerated pus in the mastoid a natural outlet, which in most cases of the kind relieves the pain. But even this was only obtained after four months. The infiltration of the lungs had so far advanced that no complete recovery could be expected, but enough cases of this kind are recorded in which after curing the affection of the bone the destructive process in the lungs came to a standstill, and even cases in which complete recovery took place, as already mentioned in case

No. 8. In the second operation I was guided by general surgical rules; we had to establish communication with the middle ear for thorough cleansing. This was obtained by the second operation. The third operation needs only of some explanation as to the formation of the abcess as far back from the primary source. At the time of its formation almost the whole external plate of the mastoid appendix had been removed. In the healing of this wound, according to my opinion, very little or no bone substance was formed (see Case 6); the external integuments, cutis, and periosteum, which were only detached and retracted at the first operation, gradually extended down into the cavity of the bone, forming a funnel-shaped opening. Thus a nail $3\frac{1}{2}$ *ctm* long, which was used, practically was only 2 *ctm*, or a little more, in the bone itself, or I may say in the neck of the funnel. In order to hold this nail in position when the external opening has such dimensions, it must be curved to adapt itself to the posterior surface of the depression, and have besides a little external plate, almost at right angle, adapting itself to the side of the head. This posterior surface of the funnel remained sore by the constant irritation of the nail, as well as by being always moist with pus, and it sometimes happened that pus found its way at this spot between the external plate and periosteum. Generally, however, infiltration takes place and formation of fungous granulations. After the opening of the occipital abscess, the fistula ran through the soft parts until the posterior surface of mastoid was reached, in which a fresh spontaneous opening was found. Thus the occipital abscess was not produced by irritation from the nail and formation of an abscess by gravitation, but by a fresh perforation of the posterior surface of mastoid near the sutura temporo-mastoidea. Although the life of the patient was not materially prolonged, my own experience in Case 8, as well as the statements and opinions of others more experienced than myself, leads me to the opinion that the whole surgical interference was justifiable.

CASE 12.—**Acute Suppuration of Left Middle Ear; Empyema of Antrum; Operation; Cure in Six Weeks.**

M———, male, twenty-one years old, consulted me in the German Hospital, June 25, 1886. He related the following history :

In May he contracted a severe cold, which, a few days later, was followed by severe pain in the left ear. He applied to a specialist in this city, who treated him with leeches and poultices without any benefit. A few days later suppuration set in and the pain decreased. The poultices were then set aside and the meatus filled daily with boric powder by the doctor himself, without previously syringing the meatus. This treatment was continued for several days, but, getting no better, he left for San José, where he used to cleanse his ear several times daily with warm water. From the very beginning of the disease he suffered intense pain ; was somewhat relieved when the discharge set in, but as soon as his ear was filled with the powder the pain became more intense, and in a few days the region behind the ear began to swell.

Status Præsens : Patient well built ; has always enjoyed the best of health ; is now mentally depressed ; his complexion pale-yellowish ; tongue coated. Copious purulent discharge from the left meatus ; after cleansing and drying its walls swelled, thus allowing only a partial view of dark-red membrana tympani. Politzer's method discovered a small perforation. The mastoid region was swollen, œdematous, and sensitive to pressure. Temperature 38° C.

He will not submit to an operation, therefore his whole temporal and part of the occipital region is shaved and repeatedly painted with a strong tincture of iodine. Ice applied interruptedly. Keeps his bed, and twice daily, after inflation with Politzer, the ear is syringed with a 1 % solution of carbolic acid. July 10th, 11th, and 12th temperature kept at 39° C. ; there was no improvement, the discharge still abundant, mastoid region swollen and sensitive ; the ice which relieved at first failed to do so now.

July 12th.—He submitted to the operation, Drs. de Vecchi and Richter assisting. Ether narcosis. Incision, from 5 to 6 *ctm* long, through the œdematous tissues, followed by profuse parenchymatous hemorrhage. Periosteum united to the bone, but easy to detach. External plate perfectly normal. Bone very hard with but few small cells. Worked with chisel and mallet to a depth of 2 *ctm*, when the antrum was reached and found full of partly

cheesy, partly fluid pus. Then Schwartze's cylindrical scoop was used to smooth down the very compact bone. In syringing no communication was obtained, the depth from inner wall of cavity to external surface of the bone measuring 2½ *ctm.* Mastoid and wound packed with iodoform gauze. Antiseptic dressing.

July 13th.—Temperature, 38° C.; patient had a good night, no pain; bandage removed, no discharge from meatus at all. The gauze in cavity of mastoid also renewed, dressed otherwise in usual manner with 2 % solution of carbolic acid. This treatment was repeated daily until the 20th of August. The temperature increased to 39° C. to the day of the operation, then decreased gradually to be normal, five days later; his complexion became more natural, his appetite improved. At the end of the first week granulations appeared; he was attended to daily up to the 20th of August, when a lead nail, 2 *ctm* long, was inserted and held by a spring. He left the hospital on the 26th. There being no suppuration at all, the nail was left off on the 28th. By the 31st the fistula had contracted so tightly that it hardly could be seen; the dressing was free from discharge.

A few remarks seem appropriate, inasmuch as, according to statement of patient, it looks as if the first treatment was the cause of the mastoid trouble. We all know that, shortly after the introduction of boric acid by Bezold, of Munich, several authorities bitterly objected to this or any other treatment by which the meatus was filled with any medicament in the form of powder, for the reason that it prevented free outflow, produced stagnation of pus, and direct infection of the cavities of mastoid. Several articles were written pro and contra, both sides quoting cases in their behalf. We are aware that boric in solution as well as in powder, judiciously applied, is one of the best remedies we have as well in acute as in chronic otorrhœa, and the fact that so much harm has been done with it when either not carefully used or pushed to extremes, is not a reason for abandoning it. In this case, I have no doubt, the mastoid affection was produced by the packing of the external meatus with the powder. But if the attending surgeon had enlarged the perforation, inflated the ear, and

after carefully syringing with a solution of boric acid, dried it, applied a moderate quantity of the powder, and packed loosely the remainder of the meatus with absorbent cotton to act as a drain, he could not be blamed. In a similar case, which I will publish in the series of 1887, I found the meatus filled and totally obstructed with a hard substance consisting of dried cerumen, epidermis, boric powder, and pus. In this case the powder was blown into the meatus without previous inspection, when it happened to be an acute otorrhœa in an ear where ceruminosis obturans existed. The skin of the meatus is often very sensitive to carbolic acid, even to the best of Schering's redistilled, which I generally use, the less irritant being Calvert's No. 1, for internal use. When, by the chronic otorrhœa, the walls of the meatus have become sore, denuded of epidermis, nothing has proved to me better than boric powder judiciously blown to cover its surface, and a loose plug of absorbent cotton filling the entire meatus to act as a drain. This cotton plug is the method used in Halle by Prof. Schwartze, and a special forceps is required to properly introduce it.

CASE 13.—**Acute Purulent Inflammation of Right Middle Ear; Empyema Mastoidei; Operation; Cure in Seven Weeks.**

C——, male, fifty-eight years old, contracted a severe cold, end of February, 1886. From 1st to 10th of March he had severe earache on right side, followed by purulent discharge from the ears. He was first treated in the country; came to the city May 24th, and placed himself under the care of Dr. Barkan, who, after trying the usual remedies, made an incision behind the ear, about the 15th of July. I was called in consultation. The doctor kindly advised patient to go to the German Hospital and place himself under my care. July 22d the patient was admitted, having profuse otorrhœa of the right side, and slight œdema of integuments over the mastoid. He complained of spells of severe dull pain in the head. He was kept in bed, had the right temporal and occipital regions shaved, and an ice-bag applied continuously, besides syringing and bathing the meatus several times daily with a warm 2 % solution of carbolic acid; McMunn's

elixir at bedtime. This treatment was followed until July 30th, without any improvement; the pain, which was relieved by the ice, returned as soon as it was left off; the swelling of external integument had decreased; the walls of meatus were swollen, and the otorrhœa as abundant as ever before.

July 30th; Operation.—Drs. Barkan and Morse assisting. Ether narcosis; incision as usual, followed by copious hemorrhage, which was stopped by compression with sponges. Periosteum adherent; external plate of normal appearance. Chisel and mallet used, and at a depth of from 3 to 3½ *mm* pus presented itself. The external opening was enlarged to 1 *ctm* in diameter, and then the scoop was used to empty the inspissated pus from the mastoid, the interior of which was changed into a large abscess, the trabeculæ having been mostly destroyed. The bone substance forming the abscess wall bled profusely; the cavity was filled with iodoform and carbolized gauze; antiseptic dressing. No communication was obtained. He remained in bed two days, and the improvement of his general health was so rapid that it was almost impossible to persuade him to stay in the room. The first dressing, impregnated with blood, was removed the following day, Dr. Barkan being present; then every 24 hours. At the second dressing it was noticed that the discharge had greatly diminished, and in a very few days it totally disappeared, and the swelling of meatus diminished. Forty-eight hours after the operation the patient's appetite and sleep increased, and no more pain was complained of. In very few days the whole cavity began to granulate. He remained in the hospital until August 23d, when a lead nail, 2½ *ctm* long was inserted. He was then treated as an outdoor patient.

August 25th and *26th.*—The nail was shortened; beginning of September left off entirely. The fistula contracted, but was cleansed every second day with a fine cotton-holder and a 5 % solution of carbolic, until September 24th, when he was dismissed perfectly cured. External meatus normal, memb. tympani thickened, and $h = \frac{10}{500}$.

September 24th, 1888, patient made a friendly visit, being in perfect health, having had no further trouble from his ear. Memb. tympani slightly thickened; $h = \frac{10}{500}$. On mastoid two verticle linear scars, 4 *ctm* long: one 1 *ctm* from posterior insertion of auricle, having in its centre a slight depression; the second, about 2½ *ctm* distant from the first.

CASE 14.—**Acute Inflammation of Middle Ear with Periostitis and Mastoiditis; Operation; Cure in Four Weeks.**

John Y——, aged 39, consulted me August 30, 1886. The fourth of July he went sea-bathing in Santa Cruz. On the fifth he had severe earache on right side. He applied poultices, and the ninth day he consulted a specialist, who lanced the ear, from which time on he had purulent discharge. After a week's treatment he noticed a swelling behind the ear. The doctor being absent, he was advised by a physician whom he consulted to come to me. All the time he had suffered excruciating pain, having sleepless nights, loss of appetite, and felt very weak.

A shoemaker by trade he had always enjoyed the best of health; he is a small, heavy-set man. His face wears signs of mental depression. The whole side of his head is œdematous; the whole temporal and especially the mastoid region, very much swollen, of dark livid-red color; temperature raised; pulse exceedingly hard and tense. I advised him to go to the German Hospital, where he was admitted on the 31st of August, and operated on the 1st of September.

Operation: Dr. Morse assisting. Ether narcosis. Auricle very much swollen; meatus hardly permeable from swelling of its walls; profuse purulent discharge. After the usual antiseptic precautions, I made an incision parallel to the posterior insertion of the auricle, 6 *ctm* long, down to the bone. The tissues were lardaceous; infiltrated. At once profuse parenchymatous hemorrhage set in, mixed with about one teaspoonful of subperiosteal pus. A sponge was pressed on the wound for several minutes. When the hemorrhage ceased I noticed that the periosteum was detached from the normal-looking bone. All efforts to discover a fistula in the bone proved fruitless, but I was able to pass my director easily along the posterior wall of the external meatus for about 2 *ctm* towards middle ear. *Spina supra meatum*, as well as the canals for the emissary blood-vessels, well marked. I began to chisel in this place, and after I had penetrated a very compact external plate 4 or 5 *mm* thick, pus began to ooze out, the external opening was made 8-9 *mm* broad, and the interior of the bone thoroughly scooped out. The bone was very firm in every direction. In syringing no communication was obtained. The meatus was well disinfected, and iodoform dressing applied.

September 2d.—Patient had a splendid night, feels refreshed, no fever, has a clearer complexion, likes to talk. In changing the dressing which was saturated with blood but dry, patient fainted. Found swelling of head very much decreased, hardly any discharge from meatus.

September 3d.—Feels so well that he speaks of going home. About same condition as yesterday with some decrease of swelling.

September 4th.—Was allowed to get up. No more discharge from meatus.

September 5th to 10th.—Remained in the hospital; swelling of the head further reduced, confined to the mastoid region. Swelling of walls of meatus diminishing rapidly. Wound granulating nicely. Dismissed from hospital.

From the 11th to 30th came to office, where he was attended to daily. The fistula in the bone was rapidly filling up with granulations, no nail being required, and the lips of external wound had to be drawn together by adhesive plaster. On the 30th he was dismissed cured. Meatus of normal calibre; memb. tympani thickened, slightly injected; neither perforation nor râles upon catheterization. Hearing, $h = \frac{100}{100}$.

The peculiarity in this case is the purulent periostitis combined with the purulent affection of the air cells. It remains to be decided whether the two processes formed independently of each other, or whether the subperiosteal abscess was directly connected with the purulent effusion in the air cells or middle ear. We often observe in inflammation of the middle ear, mostly of a purulent but often also of a purely catarrhal nature, a primary suppurative periostitis mastoidei with formation of a small abcess. In catarrhal cases the explanation of the formation of the subperiosteal abscess is somewhat difficult, unless we believe in a transmission by the blood-vessels.

In the purulent forms we almost always find that the walls of the meatus, especially the upper one, are collapsed, and if we have the opportunity of an examination as I had in this case, a direct communication will be found with the original seat of the inflammation. The cases where pus was evacuated on incision of the collapsed upper wall, are well known.

CASE 15.—**Acute Purulent Inflammation of Right Middle Ear; Caries of Mastoid; Spontaneous Perforation; Pyæmia; Operation during Comatose Condition; Death.**

Boy, H—— b—r, 14 months old. Sept. 6, 1886, at 7 P.M. I saw the child in consultation with Drs. L. C. Lane, Livingston, and S. Kahn. We learned from the mother that six weeks previously the robust and well nourished child had been taken sick with fever and treated to date by a homeœpathic physician. The condition of the child became gradually more alarming and there seemed to have been no decided diagnosis until two weeks before, when a swelling was observed behind the right ear. Some specialist was called in consultation and satisfied himself by prescribing pills to "absorb the congealed blood." The swelling kept increasing, and the condition of the child grew more and more alarming until the 2d or 3d of September, when the child became unconscious and very restless. Temperature 103° to 105°.

Status Præsens: Comatose condition; lids half open; insensibility of cornea; pupils dilated, immovable; slight attempt to cry when examined: respirations very frequent; attempts to swallow, when milk is poured between the lips; abdomen distended. Copious putrid purulent discharge from right meatus; auricle extends out at right angle; behind the same a large abscess; the skin covering it bluish, discolored, and ready to open.

Prof. Lane at once made a large incision emptying 2-3 tablespoonfuls of fetid, discolored, semi-fluid detritus. Syringed with a carbolized solution, free communication existing; antiseptic dressing.

Dr. Kahn took charge of the case during the night and recorded temperature from 103° to 105°, resorting to such internal remedies as the case might call for.

September 7th, A.M.—We met again, and after consultation I was asked to operate. The condition of the child was not materially better but no worse. With the kind assistance of the above gentlemen I operated, under ether narcosis which caused no trouble. After enlarging the incision made by Dr. Lane, we found the bone denuded of periosteum, its external surface rough; close to and at same height with upper wall of meatus a narrow fistula in external plate, into which I was able to introduce my director as a guide. I then enlarged the fistula by means of chisel and mallet, exposing the excavated mastoid appendix, which was filled with a fetid cheesy pus. I carefully scooped it, penetrating surely between 2 and 2½ *ctm;* the inner portions of the bone were soft, especially directly in-

wards, where I am convinced the internal plate was already perforated. A large quantity of carbolized solution was injected into the mastoid until the water flowing freely from the meatus had lost its fetid odor. The cavity was filled with freshly prepared iodoform gauze and an antiseptic dressing was applied.

5 P.M.—The child seemed to have rallied somewhat and I became hopeful; bandage was not disturbed. Some nourishment given with a spoon.

8th, A.M.—We met again in consultation; general condition appeared better, reacting more when handled, otherwise the same; bandage impregnated with pus, removed; thorough syringing with carbolized water; same dressing. 5 P.M.: Same general condition; bandage renewed; discharge very much decreased; not fetid.

9th, A.M.—Had a poor night. Dr. Kahn thought it would die at any time; refuses to swallow. We decided upon feeding with a soft rubber catheter. Bandage almost dry, the discharge having almost entirely ceased. Dressed in the usual manner. 5 P.M.: Coma. Same treatment. Dr. Kahn remained in care during the night, and towards morning of the 10th, after a copious and very fetid evacuation, the child died.

I cannot but reason over the question asked of me by the parents: "Could you have saved that child if called upon when the swelling first presented itself behind the ear?" Let us admit that the physician in charge was a competent general practitioner. He was called to see a child who had fever and was restless, and probably never thought and had no direct cause to believe that the trouble was in the ear. This is excusable and of daily occurrence, and specialists know that often patients, adults as well as children, who pass a restless night from otalgia, upon examination do not show the slightest trace of exudation in the middle ear, not even hyperæmia of memb. tympani. In forty-four to forty-eight hours, or later still, we are astonished to find that spontaneous perforation and exudation has taken place. But there is no excuse, after otorrhœa set in, and especially after a swelling has appeared behind the ear, which has increased daily, to still postpone the surgical interference. No doubt whatever that the child could have been

saved if the operation had been performed as soon as the swelling was noticed. No post-mortem examination was allowed, even objections raised to the probing into the mastoid, but according to the depth I had penetrated and the consistency of the inner portion of the cavity we would have found direct connection with an abcess in the adjoining portions of the brain. The sudden cessation of all discharge was probably due to the lack of vitality.

CASE 16.—**Acute Otorrhœa, Right Side, with Purulent Mastoiditis; Operated Twice; Perfect Recovery in Five Months.**

E. P. H., male, thirty-five years old, consulted me September 7, 1886. Six weeks ago he had a severe earache on right side. He applied to a physician, who could detect nothing abnormal; ordered poultices, and some drops to be applied to the ear. This he continued for one week, suffering almost continually; the ear then began to discharge, but at the same time a swelling appeared in the mastoid region, extending to the whole side of the head and face. When he called he was in intense agony, for which I gave him one teaspoonful of McMunn's elixir with whisky, and sent him to the German Hospital to be ready for operation the next morning.

Status Præsens: The whole right side of the head swollen, œdematous, densely infiltrated, the nearer to the mastoid region the harder and greater the swelling, with peculiar yellowish and pale-red color. The mastoid was extremely sensitive to touch, the patient almost fainting when it was pressed upon. Auricle œdematous, standing from the head; in meatus very copious fluid purulent discharge.

Operation in German Hospital, Dr. J. Morse assisting. Ether narcosis. Patient had been shaved and well disinfected. Incision, 6 to 7 *ctm* long, through the lardaceous tissue, which was about 3 *ctm* thick. Under the detached periosteum pus was collected. In several minutes the copious parenchymatous hemorrhage was stopped by compression with sponges. After drawing aside the periosteum, the external plate was found rough and discolored in a space the size of half a dollar, in the middle of which a small fistula led into the bone. I began to operate with the gouge, but finding the external plate harder and thicker than expected, resorted to chisel and mallet. It was then easily seen that the course of the fistula was very tortuous, running through a compact exter-

nal plate of 5 to 6 *mm* in thickness. The opening was enlarged to usual size of 1 to 1½ *ctm;* the external plate was undermined, very thick, hard, and gaining in thickness towards the occipital region. The destruction of bone substance was extremely deep inward and backward; here in probing no hard bottom could be felt, in certain spots the probe running through granulations which bled profusely. I supposed that at these spots the internal plate was already missing. Besides a great deal of granulation tissue backward and inward, the contents of this large cavity were fluid pus and cheesy substance. During the whole operation the hemorrhage was very profuse, the blood being very dark. The anterior portion was well cleansed with a scoop, less so the posterior portion. In syringing, free communication existed. The cavity was packed with iodoform gauze, producing compression, and antiseptic dressing applied.

September 9th.—The whole bandage was found saturated with blood; the house surgeon had to apply a second one to use more compression. The external dressing removed, the gauze filling the cavity was found thoroughly saturated with blood. It was partially removed, plenty of fresh gauze packed over it, producing considerable pressure. There was no discharge from meatus. Ice applied over the bandage, and patient kept quiet in bed. He had a restless night, and felt faint this morning. Swelling of head and face reduced.

September 10th.—Had a good night, with profuse perspiration. No pain; bandage dry, though bloody in spots. I removed the blood and all the gauze in the meatus which had a dirty-gray color, probably from the effect of carbolic oil.

September 11th.—Splendid night, slight sweats, no pain, wishes to get up. Bandage perfectly dry, first signs of suppuration. Thorough irrigation with carbolized solution; same dressing. Swelling of head very much reduced. Will sit up a couple of hours.

September 12th.—Was dressed when I called. Every thing in perfect condition; wishes to go home. There being no more fear of hemorrhage, he was allowed to go home after treatment.

From the 13th on he came to my office, where he was treated as an out-door patient. The cavity began to granulate nicely, the external surface of external plate was covered with granulations from soft integuments. At the end of September a lead nail was applied, the treatment carried on daily until October 11th, then,

as patient went to work, only twice a week. All discharge from and communication with the middle ear had ceased. In November I saw him four times, in December not at all. Beginning of January he complained of increased discharge from mastoid, and occasional pain, backward and downward; a small pocket had formed where the pus stagnated. I sent him back to the hospital, and scraped the posterior and lower portions of cavity, removing at the same time a part of external plate downward to provide for easy outflow. He was in the hospital two days. February 1st the nail was re-introduced, and kept to the 14th, when it was removed, being yet 1 *ctm* long; the 19th the fistula was perfectly closed, and patient dismissed.

The great destruction of bone inward and backward was caused by the thickness of the external plate, and there is but little doubt that if the operation had not been performed, most likely spontaneous perforation would have taken place toward the brain. There was undoubtedly already some destruction of inner plate, as ascertained by probing; at the same time I could not be positive, for as my probe ran through granulations, producing a great loss of very dark blood, I became somewhat timid, and abstained from penetrating deeper. At first we thought that the very dark color of the blood was due to the deep narcosis, but when we saw the hemorrhage, forty-eight hours after the operation, we had reason to fear that the walls of the sinus were already eroded.

CASE 17.—**Acute Catarrhal Inflammation of Both Middle Ears, with Slight Suppuration in Left Ear; Acute Catarrhal Mastoiditis, Left Side; Operation; Recovery in 13 Weeks.**

J. W——, male, 59 years old, first seen in consultation with Dr. Barkan, August 9, 1886. I was then informed that three or four weeks before, the patient contracted a severe cold, accompanied by pain in both ears, with slight purulent discharge on the left side. He was then treated with leeches, boracic-acid inflations. He hardly heard the watch in contact on right side; *Mt* dull and thickened; feels perfectly normal. Left side, meatus very narrow by swelling of its walls, especially the upper one. A full view of *Mt* could not be obtained, what was seen appeared

dull-red and thickened. Very slight purulent secretion. Mastoid region normal; no sensitiveness upon pressure. With the catheter a few râles heard, air penetrating only on strong pressure, and not followed either by subjective relief or by improvement of hearing. Patient physically well developed, has never been seriously ill, and led a very active life. He feels mentally and bodily depressed, complaining of fulness in the head, but no special pain. The family physician, Dr. Storror, noted that Mr. W—— was "running down," had cold sweats, slight elevation of temperature at bedtime, tongue dry and coated, appetite poor, restless at night. Sulphate of quinine and other remedies were prescribed. I suggested the bathing of the ear every two hours with a warm 1 % solution of Calvert's No. 1 carbolic acid, and then a warm Priessnitz bandage applied, besides inflations with the catheter. Patient felt relieved in a couple of days, the discharge ceased entirely, and the swelling of meatus diminished. Dr. Barkan then left him in my care. I attended to him daily at his residence until the 17th; he then came to my office three times. August 20th he stated that if his general health were better, he would not think of his ear, except that the hearing had hardly improved. *Mt* could now be better inspected; it was of an even pale-red color, and thickened; no signs of perforation visible; $h = \frac{c}{500}$; air penetrates easier; no râles. He expressed the wish to go to the springs, and I even advised him to seek a warmer and more equable climate, and upon his return to go to Dr. Barkan. I never heard of him again until October 11th, when I was requested to go to see him. I found him with his right ear bandaged, from which he had been suffering for the past few days. He stated that he had been in the country a few days, but, feeling no better, and fearing more trouble with his left ear, had returned and applied to his aurist as agreed upon. Finding no improvement, he decided to let the ear trouble alone and take its own course. He had, all the time, the cold sweats and slight fever at night.

Status Præsens: Right side relapse of acute catarrh for the last few days, accompanied by pain. Hearing $h = \frac{0}{500}$; voice hardly heard unless very close and loud. *Mt* very much injected, details not well visible. Under Priessnitz bandage and inflations with Politzer or catheter twice daily he recovered in a few days. Yet his general health was very poor; he had lost considerable flesh, his complexion was livid, almost icteric, tongue coated, and had cold sweats day and night.

October 15*th.*—The left ear had not changed materially; there was no redness nor swelling of mastoid region, but great pain upon pressure at spot of selection. He was so much afraid of what he had undergone that he requested me not to touch that ear. I called his attention to the fact that probably the bone was affected; that this was the only cause of his general debility, and advised him to send for proper assistance as soon as he had the least trouble.

October 16*th.*—Early in the morning I was sent for. I found him sitting by the fire resting his head on both hands, mentally depressed, having suffered all night dull pain in the head and shooting pains in left ear. As soon as he saw me he requested me to open his skull or do any thing I wanted; he was prepared for the worst. I expressed the wish to have a surgeon in consultation and for assistance. His friend, Dr. L. C. Lane, being his choice, I went to the doctor, who advised the operation without delay.

October 17*th*, A.M.—Operation with the kind assistance of Dr. Lane; ether narcosis by Dr. Whitwell. The usual precautions of shaving and disinfecting taken, I made an incision 5–6 *ctm* long through the slightly œdematous tissues to the bone, and separated sideways the periosteum, which firmly adhered to the bone. External plate perfectly normal; *spina supra meatum* well marked as well as emissary canals. With the broad chisel and mallet I removed the external plate, which was very compact and 4 to 5 *mm* thick. I came then to a relatively softer and very vascular portion of bone. Sharp and cylindrical spoons were used in removing partly the trabecular, partly the spongy and swelled degenerated bluish-looking mucous membrane. The mastoid of large size was very rich in cells but none of considerable size not even what was supposed to be the antrum at a depth of almost 3 *ctm.* Hemorrhage from the bone was copious, and no pus was observed. The cavity was filled with iodoform gauze; antiseptic dressing. 6 P.M.: Has been restless, and experienced pain in the head, probably from the wound.

October 18*th.*—Restless night, some pain, cold sweats, and slight elevation of temperature. The whole family noticed already this morning that he had a clearer complexion and his eyes were brighter. His neck and head felt sore. He asked if after a while he could take some breakfast. Tea and toast were allowed. The bandage was saturated with blood, but dry. It was removed

with the exception of the gauze in the wound, which was sticking firmly to the parts. The wound was thoroughly syringed with a tepid 2 % solution of carbolic acid until it appeared clean and disinfected, then external dressing applied in usual manner. P.M.: Had a fair day, slept some, complained only of soreness of neck.

October 19*th*, A.M.—Had the best night he ever had for months, no fever, felt and looked like a different man, asks for a more substantial breakfast, and inquired if some grouse that was sent to him would be an appropriate dinner. Dressing perfectly dry, removed, as well as some of the gauze in wound; dressed again in same manner.

October 20*th*.—Wound began to suppurate, patient got up and sat on a chair for treatment. He again began to take interest and inquired about his business. All the gauze in the bone was removed and renewed.

I have given the above details for a couple of days to show what a wonderfully beneficial effect the operation had in a few hours; Dr. Lane himself, our experienced and renowned surgeon, expressing himself that he had never seen such a remarkable change after an operation. From date of operation to the the 23d I visited and dressed patient twice daily, then once daily till November 3d. The whole cavity soon began to granulate rapidly, compelling me, Nov. 4th, to apply a lead nail over 2½ *ctm* long. From now on he attended to his business; he was treated in my office daily until January 21st. In December no material change was made in the nail, but in January it was rapidly reduced in size, and about the middle of the month left out entirely. The fistula contracted and was cleansed at first daily and then every second day with a fine cotton holder and 5 % carbolic solution, the holder penetrating from 1 to 1½ *ctm.* deep. End of January dismissed him cured with hearing power $h = \frac{1}{500}$. No communication was ever obtained. In October, 1887, he came to see me on private business. I found: R side, $h = \frac{100}{500}$; L, $h = \frac{5}{500}$. From the cicatrix a vertical calloid has formed, 0.03 long by 0.015 wide and 0.005 thick; its upper end is broad and gradually declining, ends pointedly downward in a depression of the external plate of about 0.01 in depth.

A very remarkable feature in this case is the lack of local pain during the whole affection until the night of the 15th

of October. There is no doubt that the catarrhal affection in left mastoid existed already at the time of our first consultation, as was indicated by the condition of his general health, though we must admit that the infection of the blood with pathological secretion or products cannot be explained in a case of catarrhal inflammation of the mastoid cells as well as in cases of caries or empyema. In the catarrhal affections one would expect more symptoms of irritation of the meninges from congestion, which were totally absent in our case. The peculiar livid, almost icteric, color of the skin, the loss of appetite, the coated tongue, and slight fever, so characteristic for all affections of the mastoid, show to satisfaction that they are produced by a slight and slow degree of septicæmia. This is proven by the wonderful improvement following every operation. Far from placing my patients under restricted diet, I allow them, from the second day of the operation, all they wish to eat, with wine or liquor if accustomed to it. Few patients are kept in the house longer than one week; being well bandaged, they are allowed to go out as soon as possible.

CASE 18.—**Acute Purulent Inflammation of Right Middle Ear; Periostitis Mastoidei with Abscess; Acute Catarrhal Inflammation of Air-Cells, with Swelling and Degeneration of Mucous Membrane; Operation; Recovery in Four Weeks.**

Miss M., æt. twenty-two, was recommended to me by Dr. James Simpson, her family physician. The first days of October she contracted a severe cold, and a couple of days later had catarrh on right side. Failing to improve under the usual care and treatment, she was advised to apply to me. I found externally auricle normal, mastoid region slightly œdematous and sensitive to pressure, meatus contracted, greenish purulent secretion in moderate quantity. No good view of *Mt* could be obtained; as far as visible the *Mt* was injected, and had a pulsating perforation in its lower portion. She had constant dull pain, with spells of shooting pain, the last especially during the night, when temperature rose slightly. I confined her to bed; after cleansing the ear I applied moist Priessnitz dressing, and every three hours ear baths with

warm water; McMunn's elixir of opium at night. This treatment was followed without any material improvement until October 24th, when the swelling in mastoid region had increased sufficiently to give fluctuation, while the subjective symptoms had remained about the same.

October 25th.—Operation, with the kind assistance of Dr. Blakie, of England, as a guest, and Dr. W. Kerr, of this city. Ether narcosis; incision in usual way, emptying half a teaspoonful of pus from subperiosteal abscess; naturally the periosteum was detached from the bone, and with a slightly curved probe I could penetrate as far as the middle ear, showing that the soft integuments of posterior wall of external meatus were totally detached. External plate normal, about 3 *mm* in thickness, when the air-cells were reached. These were totally filled with a dark-red, pulpy, gelatinous substance. The scoops were then used to a depth of about 2 *ctm*, when the antrum was reached. No more pus was found and no communication obtained. Dressed in usual manner. The following night was the first good one without an opiate; the slight elevation of temperature in the evening subsided. Already at first change of dressing the suppuration from meatus was very slight, and kept decreasing every day, and at the end of the week meatus was perfectly dry, its walls having a normal appearance. *Mt* closed, uniformly injected, pale-red in color. The wound as well as the cavity in mastoid was granulating, and at the end of the fourth week the healing process was at an end. No nail was applied, and the dressing was changed once daily. At the end of six weeks hearing $h = \frac{1}{600}$ by slightly thickened *Mt* and a hardly retracted linear cicatrix on mastoid.

This case shows that the subperiosteal abscess was in direct communication with the middle ear, as probably most of these abscesses are. The location of the pus as well as the normal appearance of the external plate might have induced a good many to desist from chiselling open the bone, and this is the course general practitioners follow. As a rule, I also admit that it is the best plan for relief, and in a few days it can easily be decided whether or not further surgical interference is needed, thus giving time to call in the specialist.

Case 19.—**Chronic Otorrhœa Left Side; Cholesteatoma Mastoidei Followed by Suppuration; Spontaneous Per-**

foration under Periosteum; Operation; Almost Complete Recovery.

Fred. D——, male, nine years old, from San José, was first brought to me June 9, 1886, with chronic fetid otorrhœa left side. After cleansing I found *Mt* totally destroyed, the middle ear filled with dried epidermoid substance, after the removal of which a small polypus was noticed filling the upper portion of the cavum tympani. With catheter the air passed freely. The polypus was removed with the snare and the child sent home with directions to syringe the ear daily with a saturated solution of boracic acid. No pain spontaneously or upon pressure on mastoid process. November 16th the child was brought back. The ear had been attended to as directed, but the discharge had not decreased; the general health was poor, and the attending physician treated the patient for malaria. The past few days the mother saw a swelling behind the left ear, which had been noticed once before when the ear stopped running, but it disappeared as soon as the discharge had returned.

Status Præsens: Pale-yellow complexion, tongue coated, temperature 38°, from the left ear fetid purulent discharge with accumulation of epidermoid masses. After removing them by syringing and forceps I found *Mt* totally destroyed, but no granulations. Auricle standing almost at right angle; behind the same the whole region of mastoid extending upwards in the temporal and backwards in the occipital region very much swelled; the skin of purple color, sensitive to touch, and fluctuating. We agreed to operate the next morning. The same evening the mother came to see me, stating that the ear was discharging very freely and that the swelling had decreased so much that she thought an operation could be avoided.

November 17th.—I called at the house prepared to operate. I found that the discharge had been very copious and the swelling of abscess reduced to one third.

Operation: Ether narcosis by Dr. Whitwell, Drs. de Vecchi and Olino assisting. The usual shaving and disinfecting was first done. Incision 4 to 5 *ctm* long, emptying a teaspoonful of pus. The skin was atrophied, and under it there was a quantity of granular substance. Periosteum destroyed in this vicinity, bone bare, its external surface discolored, and though no fistula was found its surface was perforated by fine holes very much like a sieve. No attention was paid to the topographical position, and as atrophy

of the external plate was expected no chisel was used. A gouge applied to the discolored spot liberated fetid pus as soon as the first particle of bone was removed. The opening was enlarged in every direction, first with the gouge as far as the atrophy of external plate allowed, then by means of chisel and mallet. An almost round opening 2 *ctm* in diameter was made, exposing the contents of the cavity, which were masses of cholesteatoma and cheesy substance irregularly arranged, packed together, surrounded by pus, and at certain points by granulations. They were carefully removed with the scoop, while an assistant kept syringing, very free communication with the meatus existing. The cavity thus laid bare was very large and surely extended further backward than the natural limits of the mastoid appendix of a child. In order to prevent stagnation of pus all of the undermined portions of the external plate that could be removed were carefully snipped off with Luer's sharp bone forceps or with the chisel, and the edges were smoothed with a gouge. The external opening was now no less than 2½ *ctm* in diameter. The hemorrhage, after scraping the contents, was apparently slight but very steady, and proceeded mostly from the bone substance, but to judge from the amount sponged off it was quite considerable. The cavity was first well lined with iodoform and packed with iodoform gauze. which was also applied to external meatus, besides antiseptic dressing. In the evening he felt well—was sleeping when I came. Finding the bandage dry and in order I did not disturb him.

November 18th, A.M.—Had a restless night, face flushed, temperature 39°. Bandage saturated with blood, but dry ; removed external dressing and a portion of the gauze filling the cavity (the gauze is cut into small stripes about 3 *ctm* long by 2 wide, well saturated with carbolized water and iodoform, and always freshly prepared). The plug of gauze in the meatus was also removed and found free from pus. Milk diet. In the evening, temperature 39.2° ; had a good day, having toys in bed ; no pain.

To avoid repeating facts of little interest to the reader I will leave out unnecessary details. After the 19th of November the temperature gradually fell ; he was kept in bed four days and then confined to his room six days longer, the dressing being renewed once in twenty-four hours. From the tenth day after the operation he was able to call at my office, where he was treated daily as an out-door patient till end of January, 1887, when he left

for home well supplied to be attended to by his father. The large cavity began to fill up with granulations, mostly from the posterior border of the soft parts; these granulations gradually worked their way into the cavity and lined it so rapidly that four weeks after the operation I had to fit in a nail 2½ *cm* long. In February he called five times; last, on the 26th, to have a larger nail inserted. His general health has improved wonderfully, yet there is always some slight purulent greenish discharge from the meatus none to speak of from the fistula; at times also small scales are syringed out. It could be ascertained by careful probing that the whole cavity, with the exception of the opening for the nail, was totally healed and nowhere undermined. During May, June, and July I gradually reduced the nail and then left it off entirely, because it had been reduced in length and size sufficiently, and that after being out several minutes the fistula had contracted so much that it was difficult to introduce it again. In each of the months of August, September, and October I saw the patient twice. The fistula had firmly closed with a certain amount of depression, as would be expected, but there was no sensitiveness upon pressure. The ear had been syringed daily; it still discharged slight greenish pus. In 1888 I saw him once, finding no material change; but on June 11th he presented himself, the discharge having increased and become fetid. I found some dried epidermoid masses in the middle ear, and after their removal small granulations, which were cauterized several days with chromic acid, and by the 16th had totally disappeared. The 23d he left for home with almost no discharge, but with the strict injunctions to syringe his ear once daily with tepid water, followed by instillations of sublimate 1:2000.

July 30th.—Upon a last visit the condition of his ear was still satisfactory.

The benefit in this case from the operation is surely incontestable, but yet the accumulation of cholesteatomatous masses in the cavum tympani and the occasional formation of granulations indicate that there is still some danger. The thorough evisceration of the mastoid and the way its cavity filled up, namely, by the healing into it of the retracted external integuments, such as periosteum and cutis, minutely watched until a fine conical nail could hardly be introduced after having been left out a few minutes, satisfy me that in this region no more danger has to be looked for. Un-

doubtedly there is still some caries, which is proved, first, by the recurrence of granulations, and then by the still fetid suppuration, no matter how slight it may be, in an ear which is daily syringed with disinfecting solutions. The case will remain under observation and be reported later on.

(*To be continued.*)

INVESTIGATIONS UPON BACTERIAL INVASION OF THE LABYRINTH IN THE COURSE OF MEASLES.

By S. MOOS, of Heidelberg.

(With eleven lithographic illustrations upon plates i.–vi., and a colored plate, table vii., of volume xviii., German edition.)

Translated by Dr. Max Toeplitz, New York.

I.—Pathologico-Anatomical Part.

I RECEIVED two petrous, with notes referring to them, from a friend not residing in Heidelberg. They were taken from a boy, three years old, who was admitted to the hospital on December 12th with morbilli. and died on December 19th.

Post mortem: Catarrhal Pneumonia.

I did not receive any notes with reference to the condition of the auditory organ during life.

Results of Examination of both Petrous Bones.

Both drum-membranes are united to the promontory, lustreless, yellow-red, handles of malleus not visible. Both tubes permeable for a probe of 1 *mm* in diameter, in appearance unchanged. The tympanic cavity is filled by a gelatinous exudation, the mucous membrane being extremely thickened to the extent of 3 *mm* in diameter, enclosing the ossicles; on the left side the changes are more marked than on the right. The gelatinous exudation consists of granu-

lar corpuscles and epithelial detritus. The microscopical examination of the mucous membrane of the labyrinthian wall gives the same result on both sides.

The Mucous Membrane of the Labyrinthian Wall. Microscopical Examination.

The deeper stratum, the periosteum proper, was hyperplastic, and penetrating largely into the neighboring bone, which thus presented a sinuous appearance. The proliferated periosteum had in places cut off the bone, so that in the centre of the hyperplastic tissue osseous discs partly roundish, partly oval, were visible.

The proliferated periosteum, devoid of cells, was in many portions covered by isolated or grouped colloid spherical bodies. The stratum covering the proliferated periosteum was distinguished by its bright appearance. A delicate reticulum of spindle-shaped cells and thin fibres of connective tissue form their basis; in the lacunæ of the reticulum numerous large yellow, homogeneous colloid bodies could be seen, isolated or in groups; also large round, oval, and pear-shaped cells. In the midst of this bright zone were small and large defects, imparting to the mucous membrane a fenestrated appearance even when seen with a magnifying glass. Portions of the mucous membrane were *extremely vascular*, but the greater portion of the vessels was thrombosed and obliterated; in some the contents were in granular disintegration, and wherever they were empty a *fatty* degeneration of the endothelial cells could be plainly demonstrated. In other places of the labyrinthian wall there existed a hemorrhagic infiltration of the mucous membrane.

Niche and Membrane of the Left Round Window.

The entire niche is hermetically filled by a large fibrinous clot, enclosing numerous red and some white blood corpuscles, an evidence of previous intense congestion. The laceration of the vessels of the destroyed mucous membrane of the posterior osseous frame of the niche could be demonstrated as the source of the hemorrhage; from this point pseudo-membranes could be traced into the centre of the

coagulated deposits. In the lower half of the membrane of the round window was a perforation, which was filled by coagulated fibrin, and was connected with a similar mass attached to the inner surface of the membrane in its entire surface of the scala tympani.

The niche of the right oval window and of the right stapes was filled partly by hypertrophied mucous membrane, partly by fibrinous exudations of the same character, as in the niche of the left round window; they were much larger, and enclosed larger groups of red blood-corpuscles, and more colloid bodies. Upon the promontory numerous lacerated blood-vessels could be seen. The limbs of the stapes were enveloped by the hypertrophied mucous membrane, which was covered by numerous granular cells. The stapedio-vestibular connection was normal. In the centre of the stapedial capitulum a portion of the bone was transformed into colloid substance (*cp.*, fig. 7); in that of the respective stapedial plate two such could be found (further mentioned later).

The histological changes in the labyrinth can be arranged *in two large classes.* The *first* class comprises changes due to a *purely mechanical condition*, coagulation of the lymph, gathering or aggregation of lymphoid cells, either isolated or in numerous groups occupying the *semicircular canals* and ampullæ, the inner cavities of the utriculus and sacculus, the right vestibule close to the free wall of the utriculus, and the cochlear turns of either petrous bone.

The results of some of these histological examinations are precisely the same as those in diphtheria, but they partially differ from these, inasmuch as the products of *retrogressive metamorphosis* of the lymphoid cells themselves and of the histological elements are perhaps less the *direct* than a *secondary consequence* of the morbific virus, owing to *thrombosis extending over the entire petrous bone.*

The *blood-vessels* of both petrous bones were tortuous and engorged. This globular stasis resulted in a general thrombosis, which extended through the entire labyrinth to the vas spirale and the capillaries of the stria vascularis. A few blood-vessels are empty. This general extension of the

thrombosis may be *explained by a more or less advanced fatty degeneration of the endothelium of the blood-vessels.*

As is well known, the blood remains fluid within the vessels as long as the epithelium is intact and the blood itself is in motion. The degenerated endothelia are probably partially detached, or perish by disintegration; and, since no regeneration takes place, the denuded rough places become the cause of coagulation and thrombosis. The thrombi *should, therefore, be regarded as autochthonic.* This condition of the endothelia corresponds closely with the observations first made by Ponfick that, in certain severe infectious diseases, the vascular endothelia undergo a fatty degeneration in great numbers, and are cast off. Ponfick[1] has demonstrated this condition in typhus recurrens in the living.

Special Condition of the Thrombi, according to their Situation.

Although the thromboses found in the blood-vessels, as stated above, may be attributed to a common cause, viz., the fatty degeneration of the endothelia of the vessels, their condition, nevertheless, presented a different appearance. In accordance with the character of the contents, *three large classes* may be distinguished.

The majority of the thrombi belong to the *first* class, comprising the smaller veins and the capillaries. Their contents consist mostly of red blood-corpuscles; the thrombus, nevertheless, does not present a red appearance; the red blood-corpuscles, being closely pressed together in consequence of the stasis, are irregular, flattened, and even polygonal. In an apparently later stage, they are markedly pale, in consequence of shrinking, defects are found, and a more or less wide and bright zone (*cp.* fig. 4) between the contents of the blood-cell and its wall. Besides there are found, mostly within the vessel, products of molecular detritus, and, if the products of disintegration are absorbed, smaller and larger defects as bright spots.

The *second* group is chiefly represented in the arterial and

[1] "Anatomische Studien über den Typhus recurrens."—*Virchow's Archiv*, vol. lx., p. 153.

venous vascular area of the *modiolus*. The thrombi are here plainly distinguished by a mostly *red*, sometimes orange, ochre-yellow, or clay-like *color*, although red thrombi are, as is well known, only met with in vessels of larger calibre. This alone should especially arrest our attention, the more so as G. Schwalbe[1] has recently studied and illustrated the anatomy of the vessels of the modiolus more thoroughly, and has also discussed them physiologically in a classical manner.

In the screw-shaped central canal of the modiolus an arterial coil is formed. Schwalbe gives an accurate description of the same in guinea-pigs, and distinguishes glomeruli arteriosi cochleæ majores, which, however, are not connected with each other, although their borders may overlap, and glomeruli arteriosi cochleæ minores. *According to Schwalbe, we have before us in this arrangement, as it were, a powerful current-breaker*, which causes an extraordinary diminution of the pressure and rapidity of the arterial blood, which enters through the cochlea and circulates within this beautiful apparatus, so well adapted to powerful resistances. This is effected (1) by the numberless windings, bendings, and loop-formations of the circulatory path, and (2) by the extraordinary elongation thus produced.[2] From the capillaries of the ligamentum spirale numerous veins develop, which run downward and thence as *radial* veins, with the upper side of the corresponding intermediary partition wall, centrally to the "*vena spiralis modioli.*"

[1] Ein Beitrag zur Kenntniss der Circulationsverhältnisse in der Gehörschnecke (Contribution to the Knowledge of the Circulatory Relations of the Cochlea). *Cp.* Beiträge zur Physiologie (Contributions to Physiology), Dedicated to *Carl Ludwig*, on his seventieth birthday, by his pupils, p. 200–220. (*G. Schwalbe* gives a description of the anatomy of the ear in his "Anatomie der Sinnesorgane," 1887, p. 289–559.—*H. K.*)

[2] Although not strictly relevant, I cannot forbear quoting the following physiologically most important sentence, *l. c.*, p. 216: "If we review the circulatory conditions just described, there results a most surprising relation of the scalæ to the arterial and venous circulation. The scala tympani is surrounded only by *venous* vessels, while the *scala vestibuli* covers within its wall the *arterial vessels*. The scala tympani, which is separated from Corti's organ only by the thin membrana basilaris, escapes, therefore, the influence of arterial pulsations; all arterial paths surround the scali vestibuli, which itself is, moreover, separated from Corti's organ by Reisner's membrane and the entire ductus cochlearis. We may call the scala vestibuli the *scala arteriarum*, but the scala tympani the *scala venarum*.

It is obvious, that if these blood-vessels in the normal state already diminish by their tortuous structure the pressure and rapidity of the blood circulating in them, these factors must increase every pathological effect which produces stasis. The effect is, therefore, that in these vessels the signs of stasis appear earliest and most intensely. This is in accordance with the result of examination.

The red or yellow-colored thrombi within the tortuous blood-vessels of the modiolus consist of more or less numerous *parietal* leucocytes, with or without nuclei, which extend in places even up to the centre of the vessel, in the centre of the vessels of red, rarely normal, mostly already disintegrating blood-corpuscles, granular products of disintegration of the latter and of the blood-discs also, now and then white or gray irregular granular plates, the whole being interlaced with fine-threaded fibrin. In the thin transverse sections fine fibrin threads may be seen passing through the entire lumen of the vessel. In the centre of the vessel we do not unfrequently find, in consequence of complete disintegration and beginning absorption of the contents, empty spaces, as in the first groups. The orange-yellow or ochre-colored character of the contents is, of course, due to a diffusion of the blood pigment, and indicates probably an early development of the thrombosis. These thrombi resemble, with reference to the contents, mostly the "*leucocyte thrombus*," produced experimentally by Zahn.

From the thrombosed vessels of this second group an emigration of blood-corpuscles takes place into the perivascular lymph-space; for we find therein red blood-corpuscles, as well as cells containing blood-corpuscles. Schwalbe, in his above-mentioned work on the cochlea of the guinea-pig, describes a circumvascular lymph-space as well around the vena spiralis modioli as around the arterial wall of the glomeruli. I can confirm his statements in this respect with regard to man, and also with reference to his description of the existence of a fine reticular nucleated small band of connective tissue within the arterial circumvascular lymph cavity.

Thrombi similar to those in the modiolus were also found in the convoluted vessels, surrounded by bone, which send their branches to the maculæ and cristæ of the sacculi and ampullæ. The vascular wall, however, is here also not in direct contiguity to the osseous wall, but is separated from it by a perivascular lymph cavity.

The third group of thrombi is found in the *Haversian canals.* These are only exceptionally normal. But even then at least the form of the blood-corpuscles is no longer normal, but of the same character as in the first group. Others present, chiefly, besides products of disintegration of the blood-corpuscles, only *threaded fibrin;* after disintegration and absorption of the blood-cells the majority presents *only the threaded fibrin,* and consequently complete *obliteration* of the small canal.

Weigert has lately published a method of staining threaded fibrin with anilin upon the slide, a method which has afforded the most beautiful results in the changes of the Haversian canals just described.[1]

Sequelæ of Vascular Thrombosis.

a.—SEQUELÆ RESP. CHANGES IN THE VESSELS THEMSELVES.

If the blood-vessel remains thrombosed for a long period the nutrition of the vascular wall must of course be interfered with on the part of the vasa vasorum. We actually find in the different regions of the labyrinth, viz., in the modiolus and the perilymphatic space of the semicircular canals and ampullæ, a fatty degeneration or a complete destruction of the wall elements and consequently a bright, homogeneous wall, devoid of epithelium or a hyaline thickening of the wall, here and there with dark, fine streaks or curved lines as the last remnants of the cellular elements (*cp.* fig. 4.); the vascular wall even may in the area of the hyaline degeneration undergo such a complete disintegration as to leave only aggregations of the brownish disintegrated contents enclosed by fragments of the disintegrated vascular wall.

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[1] *Cp.* On a New Method of Staining Fibrin and Microörganisms.—*Fortschritte der Medicin,* 1887, No. 8, p. 228 *ff.*

In the larger vessels, chiefly in the area of the arteria auditiva interna and its branches, the thrombosis leads to *necrosis of the vascular wall, to laceration, and to hemorrhages.* Such hemorrhages or blood-extravasations in the act of pigment metamorphosis were found in the trunk of both auditory nerves, also in the modiolus of the first cochlear turn (*cp.* fig. 1.); also laterally from the nervus ad sacculum and ad utriculum, and finally upon the mucous membrane of the labyrinthian wall and the niche of the round window. As far as the hemorrhages occur in the area of the acoustic nerve and its branches, the auditory nerves undergo, as already described in hemorrhagic pachymeningitis and in diphtheria, destruction and necrosis, and precisely as in the latter, accompanied by formation of granular cells, disintegration and atrophy of the nerves, and according to the size and volume of the hemorrhagic exudation we have smaller and larger defects in the nerve tracts. In the right acoustic such a defect measures $\frac{2}{5}$ *mm* in diameter. Similar but smaller defects were found on the side of the ramus sacculi (right petrous bone). We still see here and there in the defects the perineurium, deprived of its structure, occasionally studded with pigment granules in the shape of a rosary.

b.—INFLUENCE OF THE VASCURAR THROMBOSIS UPON THE INTERIOR MUSCLES OF THE EAR.

Unfortunately I have neglected to study the relations of the musculus tensor tympani in this direction, but I am able to offer information upon the influence of the thrombosis of the vessels of the stapedius muscle upon the latter and its nerves.

The Changes of the Stapedius—Partial Waxy Degeneration of the Muscle.

The respective specimens presented, even when seen with the naked eye, or with a magnifying glass, isolated bright spots or defects, reaching 1 *mm* in diameter, which only contained the empty sarcolemma tubes.

Staining with picrocarmine gave the muscular fibres, in which the transverse striation was still preserved, a beauti-

ful red color, while the altered fibres appeared greenish or green-yellow. The change begins in the centre of the muscle. Glycerine specimens, when examined with a higher power, showed that in some muscular fibres the nuclei and the transverse striation had disappeared, whilst the fibre itself appeared wider than normal, was colorless, homogeneous, gelatinous, or waxy, and, where the degenerated fibre protruded into the area of the above-mentioned defects, the gelatinous contents were already transformed into a finely granular detritus, and the fibre was collapsed and narrowed. Some fragments of the disintegrated muscular fibres were of a similar character. The sarcolemma was folded in places, otherwise normal. In the above-described defects we found an additional large number of normal sarcolemma tubes, covered in some places by groups of colloid bodies. The *nerve branches* supplying the stapedius muscle were partly in a state of disintegration of the myeline substance, partly in that of complete atrophy.

The blood-vessels in the interior of the muscle were thrombosed.

We have therefore before us a transformation of the contractile muscular substance of the primitive bundles of the stapedius, as was first described by Zenker as occurring in the larger muscles of the body after typhoid fever, in the form of waxy degeneration, and as was experimentally observed by Cohnheim [1] and Weil,[2] by the former after total ligature of the tongue, by the latter after ligature of its artery.

Finally, I will add, that the examination of the just-described specimens of the stapedius muscle gave a negative result with regard to microörganisms.

c.—INFLUENCE OF THROMBOSIS UPON THE LIGAMENTS AND THE ENDOTHELIAL CELLS OF THE SEMICIRCULAR CANALS AND THE AMPULLÆ, UPON THE SO-CALLED LABYRINTHIAN LIGAMENTS, UPON THE MEMBRANA PROPRIA, AND THE EPITHELIAL STRATUM OF THE MEMBRANOUS CANALS.

The fibrillar structure of the *ligaments* has disappeared; the fibrillæ are chiefly replaced by granules, arranged like a

[1] "Untersuchungen über die embolischen Processe."
[2] *Virchow's Archiv*, 1874, vol., lxv., p. 253.

string of pearls, which finally disintegrate; the ligaments thus become atrophied, rarefied, or perish. We therefore find in some specimens only a few narrow bands as remnants of the ligaments traversing the perilymphatic space. The membranous wall of the semicircular canals and of the ampullæ consequently loses its supports, it collapses and presents, instead of the sharp elliptical form, either the shape of a club, that of a pear, or that of a kidney, and in the extreme degree of atrophy of the ligaments the form of a horizontal paragraph mark, so as to bring the opposite wall in contact with each other. Besides, the *endothelial cells* undergo a retrogressive metamorphosis; in the earliest stage they show a dark, extremely granular appearance, or they resemble granular cells; this is also true of the lymphoid cells, which lose their nuclei prior to their complete disintegration. Finally all products of disintegration form a fine, molecular detritus, which is situated between the meshes of the remaining atrophied ligaments.

The membrana propria loses its striated character and presents a homogeneous, transparent appearance. The epithelial cells lose their nuclei, their framework frequently being alone preserved, or they disintegrate entirely. The canal is deprived of epithelium on its inner wall, exceptionally empty, or more or less filled by products of disintegration of the retrogressive metomorphosis, the material of which is furnished by the epithelial cells, as well as by the lymph of the endolymphatic space, or by the hyaline products (*cp.* fig. 3). The more or less complete filling of the endolymphatic space by such products of the retrogressive metamorphosis thus presents, in spite of atrophy of the ligaments, the collapse of the membranous wall (*cp.* fig. 3). As an accessory product of the tissue disintegration, a great many bundles of margarine needles are seen in the perilymphatic as well as in the endolymphatic space.

Results of Staining.

If we stain the above-described parts of the membranous hollow structures of the semicircular canals and ampullæ with picrocarmine, then *everything remains pale*, the mem-

brana propria becomes yellowish, and in no place does a red staining become apparent.

d.—INFLUENCE OF THROMBOSIS UPON THE PERIOSTEUM OF THE OSSEOUS SEMICIRCULAR CANALS AND THE AMPULLÆ.

The changes of the periosteum in measles differ from those in diphtheria essentially by the fact that in diphtheria the periosteum is either hyperplastic or atrophied, while in measles hyperplasia is never encountered. Moreover, the thrombosis produces throughout, in consequence of necrosis of the vessels, also a disintegration of the periosteum and a necrosis of the bone; its sharp outline is lost, and it appears sinuous, with jagged edges.

In those areas, in which a hyaline degeneration exists in the perilymphatic space, the periosteum may also take part in the hyaline transformation.

This *change of the periosteum* has a considerable *influence upon the nutrition of the adjoining osseous tissue*, producing smaller or larger defects with sinuous, ragged edges; these defects are sometimes filled by additional extremely fine and bright masses of detritus.

The osseous frame of these defects does no longer appear like connective tissue, but is bright, finely fibred, and poor in bone corpuscles. In the adjoining territories, which are free of defects, the bone corpuscles are no longer normal. Some are stunted, without processes, the majority in *fatty degeneration*, a change, apparently preceding the complete disintegration of the bone tissue, the products of which are sometimes, as stated above, encountered within the defects.

e.—INFLUENCE OF THE CHANGE OF THE HAVERSIAN CANALS UPON THE OSSEOUS TISSUE, VIZ.:

1. Influence of the obliteration;
2. Influence of the colloid degeneration of their endothelium upon the bone;
3. Influence upon the medullary substances.

1.—*Influence of the Obliterated Haversian Canals upon the Compact Bone Substance.*

The obliterated Haversian canals have throughout a finely striated character, and take with the above-mentioned *Weigert's* method of aniline staining a beautiful blue color upon the slide. If we examine a section from a territory of compact bone substance, stained with picrocarmine (fig. 5) *e. g.* from the cochlear capsule, we find in the midst of the bone tissue a larger or smaller number of unstained bright spots. These spots, resembling either narrow or wide homogeneous, gelatinous bands, are bounded by dark, mostly indented lines against the red-colored bone tissue (*cp.* fig. 5, lower fifth). These bright stripes apparently represent the basis-substance of the bone, in the act of decalcification or entirely decalcified. Occasionally one or more bone corpuscles are still seen in it, which have already lost their processes or present a spindle-like appearance. In many places the decalcified portions traverse the bone substance in a stellated manner, the latter like hazel-nuts bordering the already decalcified zones (fig. 5). We see in other places (*cp.* fig. 6) the further progress of decalcification, the bone tissue being in atrophy or disintegrating to a very fine detritus. In still other places this detritus, which is apparently extremely capable of absorption, is already absorbed. The bone appears fissured, multiple cavities develop, the bone substance is perforated like a sponge, and the different cavities are traversed by stripes or bands of the hyaline substance, appearing hyaline, which are moreover in places studded by the above-described detritus or very finely granular masses. Preserved portions of bone extend in places into the defects like islands, the bone corpuscles of which are already in the state of degeneration (*cp.* fig. 6).

2.—*Influence of the Colloid Degeneration of the Endothelium of the Haversian Canals upon the Compact Osseous Tissue* (*cp.* Fig. 7).

This mode of the osseous change was frequently found. In the earliest beginning we see a few endothelia on the

inner wall of the small canals transformed into round or oval, homogeneous, colloid bodies, and the remaining endothelia are in fatty degeneration. In an apparently later stage the greatest part of the calibre is filled by colloid bodies, and the blood-vessel, with its contents, is pushed aside; in the last stage the entire contents of the Haversian canals consist of extremely swollen colloid bodies, nothing can be seen of the blood-vessel or of the blood-corpuscles, the Haversian canal is already enlarged in the shape of a spindle, and finally colloid degeneration extends to the bone, where at its transition the Haversian canal is enlarged like a flask. But, of course, this process causes nutritive disorders, the effect of which manifests itself in the territories of bone adjoining the colloid masses by their presenting an appearance of finely striated connective tissue, in which the bone-corpuscles look like spindles, or are entirely absent.

3.—*Influence of the Obliteration of the Haversian Canals upon the Medullary Substance.*

The medullary substance is subjected either to a more or less complete necrosis,[1] in which the medullary cavity may be found entirely empty, or its contents undergo colloid metamorphosis. In the less intense degree of the process the colloid bodies lie between the meshes of the reticulum of the connective tissue; in the extreme degrees the entire medullary substance is in colloid degeneration. The mechanical and initiative influence upon the bone tissue bordering the medullary cavities may then be identical with those described sub. 2.

f.—INFLUENCE OF THROMBOSIS AND OBLITERATION OF THE HAVERSIAN CANAL UPON THE DEVELOPMENT OF THE OSSEOUS TISSUE OF THE COCHLEAR CAPSULE.

As is well known, the cochlear capsule ossifies by the mediation of cartilage. We find, however, even in adults,

[1] The necrosis of the medullary substance may have a double cause: the obliteration of Haversian canals, and the influence of a micro-organism (in reference to this see the bacteriological part).

still in the midst of compact osseous tissue, remnants of non-ossified cartilaginous territories. The obliteration of the Haversian canal was not without influence upon some of these cartilaginous territories.

In a specimen, stained with picrocarmine, 2½ *mm* medially from the mucous membrane of the promontory, an *unstained* zone, 1 *mm* long and ½ *mm* wide, was found in the cochlear capsule, consisting of cartilaginous cells in fatty degeneration, or already in a state of granular disintegration, the whole limited by normal cartilaginous tissue; the Haversian canals entering the borders of these bright spots were thrombosed. In another place, into whose boundary the likewise changed Haversian canals entered, the cartilaginous cells had undergone the *colloid metamorphosis*.

g.—INFLUENCE OF THROMBOSIS UPON CHANGES WHICH HAD EXISTED IN THE PETROUS BONE PRIOR TO THE OCCURRENCE OF MEASLES.

The above described changes in the middle ear, the hyperplasia of the mucous membrane, and the catarrhal affection of the middle ear have, I believe, preceded the measles. The thromboses of the vessels of the mucous membrane of the labyrinthian wall were brought about by the transformation of the round cells of the mucous membrane into numerous granular cells and colloid bodies, and by the partial fatty degeneration of the stroma of the connective tissue, in consequence of which the mucous membrane offered a macroscopically noticeable areolar appearance. The *complete absence* of epithelium in this region might be attributed to the thrombosis.

h.—INFLUENCE OF THROMBOSIS UPON THE NERVES.

Condition of the Nerves in the Maculæ and Cristæ.

A change of the nerves was noticeable where they ascend to the epithelium—*i. e.*, upon the connective-tissue stratum of the maculæ and cristæ. Also here a great number of the nerve fibres were still normal. The changed ones

presented either *a lustrous yellow, gelatinous*, or a *peculiar homogeneous, fatty appearance*, with preserved medullary sheath and apparently normal width, but with fatty degenerated nuclei of Schwann's sheath and atrophy of its protoplasm. This is probably the first stage of the atrophy. We have at any rate before us a *pure primary nerve atrophy, due to thrombosis.*

Condition of the Nerves between the Lamellæ of the Lamina Spiralis Ossea at their Point of Entrance into and beyond Corti's Organ.

Between the lamellæ of the lamina ossea, as well upon the surface as at the bottom, a disintegration of nerves takes place; in consequence of absorption of the products of disintegration an extensive formation of defects between the still preserved nerve fibres appears in places irregularly arranged (*cp.* fig. 8), or the nerve appears normal up to the neighborhood of the point of entrance. Hereupon the products of disintegration infiltrate the beginning of the membrana basilaris, and render its structure indiscernible (*cp.* fig 9 at 1′). Beyond the point of entrance the atrophy is usually complete. Within Corti's tunnel we can no longer distinguish an indication of the nerve fibre, and neither of the spiral nerve fibres; the fibres supplying the inner hair-cells are disintegrated immediately after entrance (*cp.* fig. 10, 11 *a*, and 13, 13). In no place are granular cells apparent. Since I succeeded in proving the occurrence of micro-organisms in the products of disintegration of the nerves between the lamellæ of the lamina ossea, I would like to explain the occurrence of necrosis of the nerves in this region as due to two factors, viz., to thrombosis and to a direct action of the micro-organisms upon the nerves.

Specially Noteworthy Results of Examination.

In the following I shall endeavor to describe specially noteworthy results of the histological examination. A repetition will be unavoidable where we are dealing in the different regions of the labyrinth with a recurrence of identical

changes in addition to those specially noticeable. As regards this, I ask the reader's indulgence in advance. For all noted changes cannot be comprehended from a general point of view, as, *e.g.*, in the discussion of the influence of thromboses upon the varied tissues of the petrous bones. Moreover, we do not present a mere enumeration of curiosities, for under those conditions the entire discussion would be valueless, but a description of conditions, which are either of general pathological interest, or, possibly, apt to give some explanation upon, as yet, unsettled normal histological points.

The Hyaline Degeneration.

Fig. 3 gives an approximate picture of this change, which is extremely variable and multiform. I will try in the following to give a complete description of the changes in the remaining specimens, but I am compelled to acknowledge that it will hardly be possible to give a full and elaborate description.

The endolymphatic space, except about $\frac{1}{4}$ to $\frac{1}{6}$ of its lumen, is densely filled up to the region of the epithelial layer by coagulated and disintegrated lymph, which no longer contains any lymphoid corpuscles. The epithelial stratum is indistinct. The last fourth to sixth of the lumen is filled toward the point of insertion of the membranous canal partly by a group of non-nucleated lymphoid bodies, partly traversed by a reticulum of hyaline fibres, which are studded toward the inner cavity by a number of hyaline spherical bodies, and thereby communicate with the filling mass.

The epithelial layer is in some places entirely absent, and, where it is still preserved, the epithelial cells are highly granular, or their framework only is visible. In other sections the epithelial layer is replaced by spherical bodies arranged like stalactites. The propria itself is mostly covered by hyaline bodies and molecular detritus, and, where it is visible, it is without fibrillar structure; in some places it is perforated: in these places the hyaline structures of the endo- and peri-lymphatic cavities communicate.

In the *perilymphatic space* we see neither ligaments nor their endothelial cells, but the entire space traversed, here by delicate, there by wider, hyaline reticula; in the meshes of the reticula lie here structures like granular cells or irregular clusters of molecular detritus and finely granular masses; there numerous gelatinous spherical bodies, or cactus- or coral-like masses, which are also hyaline in character, are situated upon the margin of the reticula. The *vessels* of the perilymphatic space are thrombosed, their contents are in many places in the act of disintegration. The cellular elements of the wall are devoid of nuclei or entirely absent, whereby the external wall appears homogeneous and transparent. The entire outer wall of the vessels may also have undergone hyaline degeneration, and assumes a rosary-like appearance from hyaline bodies, cut longitudinally and adherent to the wall. The external walls of adjoining vessels are sometimes connected by a chain of hyaline spherical bodies.

Whence is the Material of the Hyaline Degeneration Derived?

In the endolymphatic space the cells of the epithelial layer and the lymphoid corpuscles furnish the material for it. The hyaline mass may, to be sure, disintegrate again; this is proven by the connection of hyaline bodies not as yet disintegrated with the products of disintegration (*cp.* fig. 3; endolymphatic space).

In the perilymphatic space the periosteum, the membranous bands, their endothelial cells, nuclei of the stratum of connective tissue, and the lymphoid cells take part in the process. But this material alone would not suffice in order to fill the entire space with hyaline masses and reticula. We should rather assume that the hyaline degeneration has been preceded by a gathering, a conflux of lymphoid corpuscles, which have become polynucleated cells before the degeneration has taken place. This hypothesis is supported by the fact that in the portions undergoing hyaline degeneration groups of polynucleated cells are found (*cp.* fig. 3, z). All structures in the perilymphatic space furnish, together with collected lymphoid resp. polynucleated cells, the

material for the hyaline degeneration. *The entire condition is an excellent illustration of cell decay or coagulation necrosis* (*Weigert*).

Genesis of the Hyaline Degeneration.

The hyaline degeneration may be effected by the thrombosis or by a direct influence of micro-organisms or by both causes simultaneously. On account of the special relations of the micro-organisms in the territory of the hyaline degeneration, I am inclined to *ascribe the principal part in its development to the micro-organisms.* I have in no place observed their extraordinary spreading to such an extent as in the two sagittal semicircular canals, *i. e.*, in the endo- as well as in the peri-lymphatic space. They are here found as well in hyaline reticula as also upon the periosteum undergoing hyaline degeneration, along which they immigrate up to the adjoining bone; they are also found upon and within the hyaline bodies, and in quite unusual numbers in the molecular detritus. The latter condition is not surprising, since *Manfredi* also has proven in his experiments that, wherever the necrosis due to micro-organisms is mostly developed, *i. e.*, in the centre of the nodules, the micrococci are found in largest numbers, furthermore that the micro-organism is by no means destroyed by this necrosis. According to *Manfredi's* beautiful experiments (*l. c.*, p. 725), "it appears that the persistence of its vitality in the masses of the necrosed tissue is to be ascribed to its capacity to live even in the dilutest media, and in those most inappropriate for its nutrition."

Horizontal Semicircular Canal of the Right Petrous Bone.

Its *endolymphatic space* is filled by fine molecular lymph detritus, enclosing here and there non-nucleated lymph cells. The configuration of its membranous wall is no longer elliptical but pear-shaped. The epithelial stratum is disintegrated. The membrana propria can hardly be recognized, and is chiefly covered by molecular detritus.

The blood-vessels of the *perilymphatic space* are extremely tortuous and thrombosed, the contents partly shrunken,

partly disintegrated; the connecting bands either swollen or atrophied; the cellular elements in retrogressive metamorphosis; the periosteum proper partly atrophied, partly unravelled; the configuration of the adjoining bones either convex or concave. At the turn of the lateral into the medial limb, extending two *mm*, the *perilymphatic* space is altered in the following manner: From the wall of the membranous semicircular canal, opposite to the place of attachment of the canal, up to the periosteum of the osseous canal the space is filled *mostly by newly formed osseous tissue.*

Horizontal Ampulla of the Right Petrous Bone.

The blood-vessels near the point of entrance of the nerves into the crista, those of the crista itself as well as those of the perilymphatic space, are thrombosed; the wall elements of the latter are in fatty degeneration, their contents partially disintegrated; the membranous bands atrophied, their endothelial cells disintegrated. In the endolymphatic space several scattered groups of *non-nucleated cells, by which the cupula terminalis is covered throughout, and the space between this and the ampullar roof is completely filled.*

First Cochlear Turn of the Left Petrous Bone.
(*Cp.* fig. 1, pl. i. and ii.)

About the lower half of the first turn both scalæ are filled, the scala tympani completely, the scala vestibuli partly, by stratified masses, which are contiguous to the endostal periosteum of the cochlear capsule, but not blended with the latter; but they are throughout rather distinctly limited by the sharp outline of the periosteum from the osseous tissue of the cochlear capsule. The longitudinal diameter of the filling mass in the scala tympani measures about 2 *mm;* the interspace between the filling mass in the scala vestibuli toward the modiolus about $\frac{1}{2}$ *mm;* toward the ligamentum spirale about $\frac{1}{4}$ *mm.* The boundary of the filling mass in the scala vestibuli is formed toward the ligamentum spirale by Reisner's membrane. This membrane is pressed into the ductus cochlearis, which is thereby stunted.

Reisner's membrane itself is extremely thickened, its epithelium is undergoing fatty degeneration. Toward the cochlear axis the peripheral zone of the filling mass in the scala tympani which, stained with picrocarmine, remains bright, consists of non-nucleated and disintegrated lymphoid cells. The remaining stratified masses in both scalæ present already osseous transformation (*cp.* fig. 2), and were stained red with picrocarmine.

This examination resulted, as was intimated above, in an ossification of both scalæ of the first cochlear turn, *wherein the endosteal periosteum was not at all implicated*, in contradistinction to the condition found in a case of acquired deaf-mutism in the same region, which I have described, together with Steinbrügge in a former vol. of these ARCHIVES (*cp.* the respective illustration). In this case both scalæ were traversed by newly formed connective tissue and osseous trabeculæ, *issuing from the endosteal periosteum of the cochlear capsule.*

I consider the sharp limitation between the osseous tissue and the periosteum in similar results of autopsies as a criterion for deciding the question, whether we have to deal with a congenital or an acquired ossification in the cochlear scala. Politzer, *e. g.*, demonstrated at the eighth meeting of South German and Swiss aurists in Vienna, April, 1887, ten specimens taken from an unpublished case of ossification of the cochlea, which Burckhard–Merian, in Bâle, had bequeathed to him. They were taken from a person thirty-six years old, admitted to an institution for deaf-mutes in his tenth year, but it was not known whether the disease was congenital or acquired. The cochlea, especially in the basal turn, was filled by osseous tissue, which was sharply differentiated in the specimen from the osseous cochlear capsule. According to this description I consider the condition as an acquired ossification, which may have developed from an ossification of gathered lymphoid cells in consequence of coagulation of lymph in the cochlear scalæ, produced by bacterial invasion after an infectious disease.

The changes in the territory of the cochlear canal are of a

double character. One series is *purely histological*, the other *purely mechanical*. The latter are the consequences of the former, for pathological processes resulting in atrophy or in formation of products of degeneration, produce a dislocation, a changed position either of entire parts of the organ or of their histological elements. Since in these dislocations the alteration of the ligamentum spirale takes a principal part, we shall place its description in the foreground.

The Changes of the Ligamentum Spirale.

(*Cp.* figs. 8 and 11, plates iii. and iv.)

In consequence of thrombosis of all its vessels the ligamentum spirale undergoes a nutritive disturbance of its histological elements, here to a slighter, there to a greater, extent. In the slighter degrees we have to deal with an equal atrophy of the cellular elements of all three layers, whereby the entire ligament assumes a brighter appearance. In the higher degree single layers, either the outer (fig. 11) or the middle, *disintegrate completely* with the formation of larger defects (*cp.* figs. 8 and 11) occasionally leaving behind products of disintegration, especially colloid bodies (fig. 8 at 3). The outer periosteal layer thereby loses, entirely or partially, its nuclei and its densely fibrous character, it becomes loosened or disintegrates completely, the adjoining bone becomes more like connective tissue or is transformed into osteoid tissue (*cp.* fig. 11). In the middle layer hyaline streaks are formed in places (fig. 11, h). The connection of the stria vascularis with the inner layer of the ligamentum spirale is loosened, or it is entirely detached (fig. 8), with the formation of a more or less wide defect. The epithelial cells[1] of the sulcus spiralis externus (fig. 8, 5 to 6) perish; they may be replaced by mono- or poly-nucleated cells (fig. 8, 5 to 6). This infiltration, as a rule, is in the region of the stria vascularis

[1] In very thin sections I found the epithelium of the ligamentum spirale situated above the prominentia spiralis still regularly arranged, but without nuclei.

wider, more abundant, and contains many cells, which have again lost their nuclei. In consequence of the described changes, the form of the entire ligamentum spirale is extremely altered. *Cp.* in reference to this the ligamentum spirale of fig. 8 with that of fig. 1.

Reisner's membrane (*cp.* fig. 8 and 9, plates iii. and iv.). The *histological alteration* of Reisner's membrane comprises all its elements; the membrane loses, by fatty degeneration, the endothelium of the vestibular surface as well as the epithelium of that facing the ductus cochlearis, and may finally appear structureless (fig. 9, 5). At the beginning of its origin on the vestibular side of the lamina spiralis ossea, and still some distance further upward and outward, the entire membrane shrinks by disintegration (fig. 8, at 9). In an entirely structureless condition the marginal portion may be covered by coagulated lymph or molecular products of disintegration (fig. 9, 6). Its surface is in other specimens covered by lymphoid cells (its outer two thirds in fig. 8, at 9), or by molecular detritus (inner third in fig. 8). There are sometimes on its margin hyaline bodies or clusters of either non-nucleated or nucleated lymphoid cells (fig. 8 in the middle third).

The *mechanical change* in Reisner's membrane is due to two factors: the first consists in the described dislodgment of the ligamentum spirale, its atrophy, and detachment of the stria vascularis from the underlying connective tissue; the second factor of the dislocation depends upon the atrophy of Reisner's membrane at its insertion upon the labium vestibulare of the lamina spiralis ossea.

The Changes of the Membrana Corti or Tectoria.

Everybody, even with a superficial knowledge of the topography of Corti's organ, inspecting the illustrations 8, 9, and 10, will be struck by the strikingly changed position of Corti's membrane. It is, as is well known, stretched above the sulcus spiralis internus, and extends to the outer hair cells beyond Corti's organ; it covers the latter, wherefrom the name "tectoria" is derived. In all

three illustrations no tectoria is really discernible; it lies at a great distance from Corti's organ above the sulc. spir. int. (fig. 9 and 10 or fig. 8), and is dislocated so far outward as to lie just above the crista spiralis.

We do not see its profile, as in other radial sections, but it is placed in such a manner as to present its entire lower surface (fig. 8, 13; fig. 9, 10; fig. 10, 8).

It is easy to discover that this *repeatedly* observed *condition* is not artificially produced.

The Changes of the Crista Spiralis.

If we view the figures 8, etc., we miss the concavity of the sulcus spiralis internus and the sharp-edged prominence of the labium vestibulare; the normally deep furrow is flattened, and the sharp-edged prominence of the labium vestibulare is represented by a blunt protuberance, apart from its infiltration with lymphoid cells. Embryological examinations prove (*Boettcher*, *Middendorp*, *cp. Schwalbe*, "Anatomy of the Ear," p. 356), that the acute angle of the labium vestibulare has as its precursor a slightly pronounced obtuse angle, which, however, assumes the definite form already during embryonic life. The illustrated morphological changes are possibly in connection with the tension and consequent detachment of the tectoria, which is intimately connected with the crista, and has been already discussed above.

The cells situated in the interdental sulci disintegrate, as also the epithelial cells upon the surface. The cells of the sulcus spiralis internus may be preserved; but they present a granular appearance (fig. 9 and 10), or they disintegrate partially (fig. 8 and 11); in this case they sometimes increase before disintegration and assume a darker shading, a granulation of the nucleus, which extends at the expense of the protoplasma of the cell-body close to the cell-wall (*cp.* fig 11). In this process of degeneration and disintegration also the labium tympanicum of the crista may take part, whereby it assumes a furrowed or fimbriated appearance (*cp.* fig. 10 and 11). The space of the sulcus spiralis

internus may at the same time remain free, or is more or less filled by lymphoid cells, or molecular products of disintegration (fig. 10). The latter are frequently superimposed upon the labium tympanicum, which thereby appears narrowed (fig. 8).

Changes of the Membrana Basilaris.

In vertical radial sections, as is well known, two strata of different structure are distinguished. The *upper* is a homogeneous continuation of the hyaline upper plate of the labium tympanicum ; it continues on the outer side into the hyaline lining of the sulcus spiralis internus. *Schwalbe* calls this layer the *membrana basilaris propria*. Upon the lower surface a tissue layer abundant in cells, the *tympanal lining stratum* (*Retzius*), is located as continuation of the lower lamella of the labium tympanicum.

The sharply pointed transition of the labium tympanicum into the basis substance of the membrana basilaris was lacking in several specimens. It can be distinctly recognized in fig. 9 and 11; it is not visible in fig. 8 and 11. in consequence of a deposition of products of disintegration of the adjoining nerve fibres. In some specimens the propria of the basilaris in its entire course up to the sulcus spiralis internus, where it continues as crista basilaris into the ligamentum spirale, is not visible on account of being infiltrated by lymphoid or polynucleated cells (*cp.* fig. 10). The same condition frequently existed in the nucleated hyaline layer situated upon the basilaris propria.

Mechanically, the outer portion of the basilar membrane is altered, inasmuch as in consequence of the atrophy of the adjoining ligamentum spirale it loses its tension, and instead of being horizontal, appears convex toward the vestibule. (*cp.* fig. 8 and 11).

The changes of Corti's organ are characterized by hyaline degeneration, partial or complete disintegration and atrophy of its cellular structures ; the nuclei remain longer preserved than the cell-body. A certain regularity in the succession of time of the disintegration can be distinctly recognized.

The inner epithelial cells perish earliest, followed by Corti's and Deiters' cells, then succeed Corti's arches, viz. the inner before the outer, and finally the lamina reticularis. The outer supporting cells resist longest, longer than Claudius' and Boettcher's cells. The various structures of Corti's organ still remaining, hereby frequently change their position, a dislodgment here in vertical, there in horizontal direction ; the nuclei are dislocated to places where they do not exist under normal conditions. The appearance of the atrophy in the different specimens are extremely multiform and varying, and its condition found in all specimens can hardly be described in all its details. A number of them is represented in fig. 8 to 11 and their exact description can be found in the explanation of the illustrations. It is, however, necessary to discuss the question, whether we have really to deal with atrophic changes or artificial products respecting the action of reagents.

"In reference to the rods," *Boettcher* says, "when it was still disputed, whether they were easily destructible or very resistible structures, to whose substance Henle ascribed even a hardness approaching that of cartilage, I have pointed out their difference in fresh specimens from those prepared with muriatic or chromic acid. They obtain their great resistibility from the action of these acids and can then be easier studied in certain respects." Since our specimens have been decalcified with chromic acid, the objection, that the illustrated *anomalies of the connection* of the rods are artificial and not pathological, loses its force. I believe upon the whole, that it would lay the facts under restraint, if we consider the described changes of Corti's organ as due to the action of decalcifying fluids. These can by no means produce a complete atrophy of the inner supporting and hair cells. I do not hesitate to put the described changes in relation to the thromboses in the ligamentum spirale, especially to the described alteration of the stria vascularis.

"We believe," *Schwalbe* says, "to recognize in the stria vascularis the organ which serves for the secretion of lymph." ("Organ of Hearing," p. 353). If this is, as in our case (*cp.* fig. 8 and 11), destroyed or degenerated, there

results a fundamental condition for the alteration of the lymph and for nutritive derangement in Corti's organ with its sequelæ.

Applications of the Results of the Examinations to Controversies upon the Normal Histological Structure of Corti's Organ.

Boettcher in his above-mentioned "retrospects," etc., has added to his own observation a full criticism upon the most important, still pending controversies upon the structure of the cochlea.

It is not within the province of my subject to enter into a discussion upon these. I may have another opportunity to make use of my specimens with especial reference to this. I shall at any rate point out to experts, that specimens presenting atrophic processes in Corti's organs are especially appropriate to decide certain points of controversy.

With reference to two points in dispute I concur entirely with Boettcher: first, that Corti's organ presents a third zone, the so-called *marginal cord* (*cp.* fig. 9 and 10) and furthermore, *that Corti's cells show a basal process.* This latter condition can undoubtedly be recognized in various specimens of mine. It is distinctly illustrated in fig. 9 and especially at *c* in fig. 11.

II.—Bacteriological Part.

General Literature and History.

E. Klebs[1] raises, in his "General Pathology," on p. 336, the following question: Are there any processes which can with sufficient reason be considered as produced by coccacea, and which, on account of their peculiarities, form a group of the same category?

If we answer this question, we should, according to Klebs, first consider the *acute exanthemata*, which have always been comprised in one group, on account of important affinities in the manner of their extension and

[1] General Pathology or the Study of the Causes and the Nature of the Processes of Disease." First Part: The Causes of Disease; General Pathological Etiology. Jena, 1887.

transmission, as well as in their clinical symptoms. After an explicit explanation of this sentence, he continues as follows: "This typical course, which manifests itself here more distinctly than in any other infectious disease, has brought to light the homogeneousness of the forms of disease, when nobody dared to dream of their parasitary nature. Now we may confidently form the postulate, that all these forms of disease *are produced by microörganisms of similar character.* They all have in common the epithelium as a suitable nutrient soil, and cause from this focus those local and general symptoms in cyclical succession. According to our present, although incomplete, experience, it cannot be doubted that they may be classed among the coccacea. This is sure in *variola*, doubtful in *scarlatina* and *measles.*" Klebs has himself demonstrated that in variola the cocci show special arrangement, which he considers as an important characteristic, viz., the *arrangement in fours.*[1]

These *tetracocci* occur, besides isolated cocci, and those which lie together in irregular clusters, in the vaccine lymph as well as in the secretions of the mucous membranes of the pharynx and trachea in variola, and also in the vaccine lymph of man and cows. Cornil and Babés have also demonstrated the tetracocci in variola and in vaccine pustules.[2]

With regard to measles, full bacteriological examinations have been made of late by Cornil and Babés.

Cornil and Babés found in lungs affected with pneumonia, of patients who have died of measles, large numbers of diplococci, which, similar to the gonococci are composed, of two crescentic bodies, separated by a bright intermediary substance, form large zoöglœa masses or are arranged

[1] "In view of the wide distribution of tetracocci-forms, and especially in consideration of the fact that according to Bordone-Uffreduzzi, such forms are not missing among the microphytes found in the normal skin, the specific significance of the so-called tetracoccus variolæ (Klebs) seems to be quite doubtful," etc. "Text-book of Pathological Mycology," by P. Baumgarten. Second half, p. 389.

[2] With regard to the presence of micrococci in human variola, the first statements have been made, according to Klebs, by Luginbühl, who considered the giant cells, demonstrated by him in the first beginning of the pustule, as bearers of the virus.

in chains. Babés' cultures with blood from the exanthema of measles, and also products of inflammation of the lung, pleura, lymph-glands (upon human and bovine blood serum), resulted in the formation of streptococci. Their members were 8-shaped micrococci, but some were also found resembling streptococcus pyogenes. Subcutaneously injected into the skin of the nose of young guinea-pigs, they cause redness of skin, fever, and sero-purulent conjunctivitis, changes produced in the same manner by direct transmission of the blood of a child affected with measles. (*Cp.* Klebs, *l. c.*)

L. Manfredi has published of late a lengthy paper on the bacterium of measles.[1]

In two cases of pneumonia following morbilli, the sputa were shown to contain Friedlaender's pneumonia cocci upon plate cultures. Manfredi found an additional coccus pathogenic to animals, which produced, in experiments made upon animals, a new typical disease, which induced the author to name the respect. microörganism "the micrococcus of the progressive lymphoma or granuloma in the animal body."

The shape of this microörganism is oblong, with rounded or obtuse ends. They are found chiefly single or as diplococci, sometimes more as bacillar elements. They stain readily without characteristic features. After subcutaneous injection, multiple granular neoplasms develop in form of nodules in the inner organs. In the resp. tumors the cocci are found most numerous in the centre of the cells, less frequently outside the cells, rarely in the blood-vessels; they have, therefore, a chiefly intracellular action. The lymph-vessels are the path of infection.

Own Observations. General Remarks.

From the former description of the changes found in the petrous bones, the fact of their almost complete accordance becomes apparent, a fact of great importance for the sub-

[1] Ueber einen neuen Mikrokokkus als pathogenes Agens bei infectiösen Tumoren. Seine Beziehungen zur Pneumonie. (On a new micrococcus as pathogenic agent in infectious tumors, and its relations to pneumonia.) *Fortschritte der Medicin*, 1886, No. 22.

sequent remarks. The readers, nevertheless, will be surprised, some even disappointed, that I did not succeed in proving the existence of microörganisms in a decisive manner in the *different* parts of the tissues, except in one of the two petrous bones first examined, *i. e.*, in the left. In the right I could demonstrate[1] the resp. microörganism only in the medullary cavities and in the products of disintegration of the nerves between the strata of the lamina ossea. It would be wrong, however, hence to conclude that it had been the principal factor of the changes only in the *left* bone. If some bacteriologists admit that the occurrence of microörganisms is sometimes not easily proven, even in experiments made on animals, this holds good the more for the resp. examinations on pathological specimens, and especially for those on the human petrous bone, which must be decalcified for weeks before it can be cut and the sections be bacteriologically examined. And then the investigator must be so fortunate as to succeed in making very thin sections, else the bacteriological examination is not practicable.

In addition, it is difficult to demonstrate the microörganisms in the tissues, especially in some territories of the labyrinth we have to deal with very small numbers of microörganisms, or the changes are old. We find, as is well known, microörganisms sometimes only in freshly diseased parts, *e. g.*, in erysipelas (Koch, Fehleisen), or the difficulty of their demonstration lies in the microörganism itself, the life of which may be only of short duration, it may degenerate quickly, etc.

Methods of Examination.

The examinations were made on sections, viz.:

(*a*) According to Manfredi's method, *l. c.*, p. 724, weak watery solutions of methylene blue. The sections remain in it many hours (it is not stated how many), are washed afterward in distilled water and treated with alcohol. Then clearing up in turpentine oil and preserving in balsam.

[1] In the two petrous bones of the second case, disfigured in consequence of too extreme decalcification, I succeeded in proving the existence of microörganisms, in the medullary cavities and in the membranous semicircular canals in hyaline degeneration of one side.

(*b*) The well-known *Gram's method*, also recommended by Manfredi, had furnished me with beautiful results, which were more distinct than those obtained with the former, because the sections were better decolorized. The resp. sections, preserved in xylol Canada balsam, furnished lasting specimens.[1]

(*c*) According to Gabbett's method of staining tubercle bacilli.

Gabbett's method of staining tubercle bacilli in slide specimens is a slight modification of that of Neelsen.[2] The staining solution, which, according to Gabbett, is superior to all other aniline staining fluids, is prepared as follows :

Solution I.—One part magenta red (English name of fuchsin), 100 parts of water, 5 per cent. carbolic water, 10 parts of absol. alcohol.

Solution II.—100 parts of a 25-per-cent. solution of sulphuric acid, 1–2 parts (grammes) methylene blue (the solution must be of a dark-blue color).

For the preparation of slide specimens solution I. is slightly warmed in a watch-glass until vapors begin to rise. The slides prepared as usual swim *two minutes* upon it.

The treatment with acids and the contrasting after coloring is now combined cleverly *in one act* by the use of solution II.

The slide is washed off quickly in water after use of solution I. and placed in solution II. for one minute. It remains only to wash in water, to dry event., and to preserve in Canada balsam in order to finish the specimen.

So far H. S. Gabbett.

Dr. Ernst gives the following results :

(1) Slide specimens, as well as sections, are beautifully stained in solution I. (for two minutes), and solution II. (for one minute).

[1] "The microscopical examination of the micrococcus in tissue sections is different in some respects, which, I believe, should be ascribed to two reasons : First, that this micrococcus retains the aniline colors only slightly after a certain period of its existence ; we can, therefore, after staining of the sections, not determine beforehand the means of decoloration for the tissue, and we run the risk of decolorizing also the parasites. Secondly, that the mentioned microörganism, which is rarely arranged in zoöglœa form, and, as a rule, rather attacks the protoplasm of the cells, whereby the nucleus is sacrificed by active segmentation and fragmentation, remains frequently exhausted in the midst of the not completely decolorized fragments of the nucleus."—Manfredi, *l. c.*

[2] *Cp.* according to Ernst, a review published in the *Medical Record*, July 9, 1887, concerning a note in No. 3,319 of the *Lancet*, April 9, 1887.

(2) It is unnecessary to *warm* the solution, a very desirable consideration for celloidin sections.

(3) Decolorization of the tissue and cells is complete to a degree never attained before, except by means of Futterer's modification (quite a tedious procedure); the contrast color thereby becomes much neater and cleaner.

(4) The staining fluids, as well as the colors in the Canada-balsam specimens (both in sections and in dry specimens), have remained unchanged in composition and intensity for three months.

(5) The single colored bacilli are brought out with great distinctness. The discovery of isolated bacilli, especially in the tissues, is extremely facilitated, probably because no red color whatever is left in the tissue.

From my own experience I can recommend this method as a very convenient and reliable one for sections of tissue containing cocci.

The form, arrangement, and size of the microörganisms, conditions and paths of invasion. (*Cp.* the colored plate.)

The microörganism in question should be ranked among the *coccacea*. It differs morphologically from that described by Manfredi by the constancy of the *round* form, *the spherical growth, while Manfredi describes his own as oblong, with rounded or obtuse ends;* the round form is constant, whether the parasite occurs as mono-, diplo-, and tetra-coccus (rarely [1]) in groups or irregular clusters, in chains or in strings. *The chain form* is, upon the whole, the *prevailing* one. The size of the single cocci varies, the larger ones being more numerous. If we place, morphologically, much value upon the chain formation, we should call this parasite also a streptococcus. *But it is very doubtful whether it may be considered as the specific microörganism of measles.* With reference to this point extensive investigations are necessary. I would like, however, to emphasize one point. In bacterial invasion of the labyrinth after diphtheria we have also to deal with a streptococcus and, as we know, with an *accidental and not with a specific microörganism* of diphtheria. But we know in this disease the conditions and paths of in-

[1] This rare occurrence as tetracoccus must be strongly emphasized with reference to the above-mentioned statement of *Kleb's* concerning the tetracoccus in variola.

vasion, to wit, loss of the epithelium of the mucous membrane of the pharynx and the air passages, penetration into the lymph-paths, chiefly after development of tissue necrosis in the pharynx and then from the lymph-paths into the blood. If we now suppose that the streptococcus found in measles in the labyrinth is identical with that in diphtheria, therefore but *an accidental and not a specific microörganism*, the condition should be considered a complication of this infectious disease (of measles), like in various other diseases which are associated with affections of mucous membranes, as in variola, typhoid and puerperal fever. *Lesions* of mucous membranes, however, are in measles less frequent and less marked than in the just-mentioned infectious diseases; acute forms of pharyngitis and tonsillitis occur, to be sure, but, as far as I know, no necrotic destructions. Thus it only remains to assume an immigration of the streptococci into the lymph-path and thence into the blood, by means of a lesion of the epithelium of the mucous membrane developed from the catarrhal affection of the pharynx, tonsils, and air passages. The conditions of invasion are, at any rate in measles, more unfavorable for the streptococcus than in the other infectious diseases; the danger of a bacterial invasion of the labyrinth in this infectious disease is, therefore, much smaller. Affections of the labyrinth in measles, are, in fact, rarely (*cp.* the clinical part) observed. In my practice, extending over almost 30 years, I met with only the two cases described in the clinical part. The possibility of a more frequent occurrence, however, in autopsies, is by no means hereby excluded. If we consider that, as far as I know, the labyrinth of persons who died of measles had never before been carefully examined, and that my examination is the first of the kind, it would be a strange coincidence if I should find this extremely rare exception in the very first examination.

Regionary Occurrence.

The efforts to discover the microörganism in the different parts of the petrous bone proved frequently fruitless, especially in the right petrous bone, in which I succeeded in

finding it only in the medullary spaces and in the products of disintegration of the nerves, between the strata of the lamina spiralis ossea. On the other hand I found it in the left petrous bone, not only in this, but also in the Haversian canals, in the endo- and peri-lymphatic cavity of the semicircular canals, and especially in the region of the hyaline degeneration, and here again in large masses in among the hyaline products of disintegration. Furthermore, it was seen in the periosteum in hyaline degeneration, and along this up into the necrosed osseous parts described above, especially in their sinuses. I could not prove in the above-mentioned territories their intracellular occurrence, except in the medullary cells. I could count them here up to six, arranged in chain-form at the periphery of the medullary cell, near the cellular wall. They could also be demonstrated within the colloid spherical bodies of the medullary cavity. The results of examination in the cochlea were negative.

Biological Properties of the Microörganism.

After it has been found impossible to distinguish *morphologically* the microörganism found in measles from that observed in diphtheria in the labyrinth, we will try to prove this *biologically*. This consideration appears the more urgent inasmuch as there exist, as is well known, five different kinds of streptococci, which cannot morphologically be distinguished from each other, but the pathogenic actions of which nevertheless differ in many respects. If we now compare the respective histological results of examination of the labyrinth in diphtheria with those obtained in measles, we record, above all, the important fact that we found *in the former signs of new-formation*, viz., small-celled infiltration, *e. g.*, in the Haversian canals and the medullary cavities, and hyperplasia of the periosteum in different regions, *e. g.*, in the semicircular canals, in the periosteum of the pyramid of the petrous bone, *as well as necrosis, e. g.*, of the bone and the medullary substance, *while the examinations in measles demonstrated nowhere signs of new-formation, but only those of necrosis.*

Our results of examination agree with those of Manfredi with regard to this pathogenic action. He says (*l. c.*) that the formation of micrococcus used in his experiments appears to be a production of *cell-necrosis*, by destroying the nucleus *within the cell itself* and the structure of the cell. The proof of the penetration into the interior of the cells was brought, at least undoubtedly in the medullary cells, and the signs of necrosis in the different regions of the petrous bone were plainly visible. *The necrosis cannot, indeed, be considered everywhere a direct intracellular effect of the microörganism, but it is frequently only an indirect one,* produced by the thromboses in consequence of the mycotic fatty degeneration of the endothelium.

Paths of Degeneration in the Labyrinth.

The demonstration of the microörganism within the Haversian canals and the endolymphatic cavity proves the existence of the respective parasite within the paths of the lymph and blood. The manner in which the immigration thence into the labyrinth takes place may be learned from my investigations on bacterial invasion of the labyrinth after diphtheria, which explain the different possibilities on the basis of the special anatomical and histological relations of the labyrinth, particularly as regards the connection of the endolymphatic and perilymphatic spaces with those of the cranial cavity.

Brief Review of the Genesis of all Discovered Changes.

I consider in measles, as well as in diphtheria, the coagulation of the lymph a *purely mechanical condition*, due to the immigration of the microörganism. The disintegration of the lymph elements in the endolymphatic space, which is without blood-vessels, may depend upon an intracellular action of the parasite, and may then be considered as a mycotic necrosis. Their disintegration in all tissues containing blood-vessels may be effected by the same cause or may be the consequence of the thromboses, or both. The thromboses are the consequences of a mycotic fatty degeneration of the endothelium. Their consequences have already

been explained. I would like to point out the fact that from the thromboses the numerous noteworthy changes in the region of the ductus cochlearis can be explained. How much here, chiefly in Corti's organ, is due to a *direct* influence of the microörganism, cannot be stated on account of the negative results of examination.

The biological action of the parasite takes probably a prominent part in the production of hyaline degeneration, and also in the disintegration of the nerves between the strata of the lamina spiralis ossea.

The necrotic degeneration, or the complete atrophy of the medullary substance of bone is probably the consequence of the obliteration of the Haversian canals and of a *direct* influence of the microörganism upon its tissue. Each factor may, moreover, produce the same result.

The vascular necrosis also may have this double cause. It may develop, as is well known, in continued thrombosis, since it affects the nutrition of the vascular wall itself, but it may be also the direct consequence of the mycotic action.

III.—Clinical Part.

From the appended literature a comparatively great infrequency of affections of the labyrinth in consequence of measles becomes apparent. They seem to be still less frequent than those in consequence of diphtheria. I have observed only two typical cases, whose full report is justified by the rareness of such observations. They are characterized, like those after diphtheria, *by permanent complete deafness, by disturbances of equilibrium, viz., staggering gait.* For the explanation of these symptoms, which are fully discussed in my paper on diphtheria, the pathologico-histological results of examination, in the labyrinth of persons who died from measles furnish sufficient points of support. I do not enter here, therefore, into an additional discussion of these conditions.

HISTORIES OF CASES.

First Case. Complete Loss of Hearing with Staggering Gait in the Course of Measles.

I am indebted to the kindness of Dr. Habermehl in Edesheim, Palatinate, for the notes of this case.

"The girl, five years of age, was seized February 12, 1885, with measles and attended for three days. Besides the usual symptoms, fever, inflammation of the organs of the chest, extensive exanthema, etc., *hardness of hearing developed from the beginning, which soon resulted in complete deafness.* Hoping that the hearing would be restored with the expiration of the disease, I delayed active treatment until March 1st. I then made instillation of astringents into the external meatus, to which Dr. Stempel in Neustadt added vesicants. The epidemic was extensive, and had upon the whole a favorable character, with a few sequelæ. This child *alone* was afflicted with deafness, which doubtless has not improved since in the least, and seems to depend on a nervous paralysis, since paralytic symptoms were manifested in the peripheral parts, as was noticed in walking. The child stumbled in walking, moved rapidly onward without being able to restrain herself. Further observations could not be made since I made three visits, and the child was brought to me three times. This is the way in country practice."

The examination, which I made May 4th, revealed a perforation of the posterior half of the right drum-membrane, without a trace of pus, either in the external meatus or in the remainder of the drum-membrane or beyond the perforation (confirmed condition !). The left drum-membrane was drawn inward and presented considerable dulness of the mucous membrane. *Absolute loss of hearing for all sounds. Marked waddling gait.* After seven unsuccessful injections of pilocarpine the mother withdrew the child from treatment, which Dr. Habermehl was requested to continue at home. I have heard no more about the further course, but I suppose the deafness remained unchanged.

Second Case. Complete Loss of the Faculty of Hearing with Remaining Staggering Gait after Measles.

M. St., girl, twelve years of age, presented herself at the aural clinic for the first time May 30, 1885.

The mother states that the girl was five weeks previously seized with fever and intense headache, followed after a week by measles, from which recovery took place without particular complications. In the second week of the disease "the hearing was quickly lost, vertigo developed, and in the third week staggering gait when leaving the bed."

The examination revealed shortening of the cone of light on

either side and dulness of the mucous membrane of the drum-head. The patient is absolutely deaf and staggers still considerably. After twelve injections of pilocarpine, which proved entirely unsuccessful, the patient withdrew from treatment.

Review of Both Cases.

With the exception of the perforation of the right drum-membrane in the first case, which perhaps developed before the attack of measles, both cases present a complete agreement of symptoms—*absolute deafness and disturbance of equilibrium, viz., staggering gait.* Both cases can be explained by means of the above-described changes of the labyrinth, to the details of which I now merely refer. The *changes in the semicircular canals, or in the ampullæ* respectively, must probably be considered the anatomical causes of the disturbances of equilibrium. The deafness could be due to *hemorrhages* into the larger nerve bundles, to destruction of the nerves, and a great number of nerve fibres, furthermore to filling of the cochlear scalæ in consequence of stratified apposition of lymph cells with their consecutive changes, and finally to the fully described changes in the terminal apparatus, among which the colloid degeneration and the atrophy of Corti's organ rank first. I need hardly point out that no treatment can influence such changes, as its failure indicated in both cases. The number of my observations, however, is too small to conclude from them that the prognosis is unfavorable in all cases of nervous deafness after measles. They belong at all events to the severest forms of nervous diseases of the ear.

F. Rohrer's remarkable observation is a proof[1] that the prognosis need not always be absolutely unfavorable.

A girl, twelve years of age, was suddenly affected with complete deafness in both ears, a week after the eruption of measles. Beginning and course of the disease did not present any extraordinary features. Subjective noises on both sides, here and there with transition into distinct musical tones; staggering gait is not recorded. The examination of the history had to be made in writing on account of the complete deafness. Absolute loss of

[1] A case of sudden deafness after measles. *Correspondenzbl. f. Schweizer Ærzte*, vol. xiv., 1889.

hearing for all sounds, even for bone-conduction. Negative result of examination; slight swelling of Eustachian tube. Deafness lasting twenty-five days. Return of the faculty of hearing after headache of two days' duration, peculiar disturbances of vision, and psychical and nervous attacks; sulkiness, melancholia, fixed look, sluggish reaction of pupils, and a series of eclamptiform attacks.

Besides a central lesion, Rohrer says in the review of the remarkable case, there existed undoubtedly a certain connection of the affection with the measles and an affection of the tube and the middle ear. A diminution of the hearing power was probably thereby started. The subsequent absolute deafness, connected with profound disturbances of the nerve functions in general and of the acoustic nerves in particular, might have been produced by central changes and lesions. Hyperæmia of the labyrinth and a neuritic irritation of the acoustic from the middle ear may occur. Rohrer mentions here the immigration of coryza bacteria (Klebs) through the frontal and ethmoidal sinuses, and also through the tympanic cavity, whereby cerebral hyperæmia, especially of the base and œdematous swelling of the arachnoidal tissue and of isolated nerve sheaths may be produced. These serous infiltrations at the basis cerebri, Rohrer thinks, are under these circumstances easily produced; *transitory* severe meningeal symptoms, principally in children, may not infrequently be diagnosticated as consequence of these occurrences. One might at all events take exception to the hypothesis of the genesis of the complete deafness on account of hyperæmia and irritation of the acoustic, because these changes hardly produce absolute deafness. It may be perhaps more fully explained by the supposition of a hemorrhagic exudation at the acoustic, absorbed again in the course of weeks. The remarkable concomitant cerebral symptoms, which were associated with the return of the hearing-power, render, moreover, this case unprecedented.

Synoposis of the Literature on Measles as Cause of Aural Diseases.

1. I. Toynbee, "Diseases of the Ear," mentions—in chapter xv., under section 3—hardness of hearing produced by the action of

morbific poisons on the nervous apparatus of the ear, besides gout, typhoid fever, scarlatina, and mumps, also the measles.

2. On aural diseases as consequence and cause of general diseases. Inaugural Dissertation, by Joh. Heydloff, Halle, 1876. Measles in relation to scarlatina are considered as comparatively benign with regard to aural affections.

3. I. Gottstein. Contributions to aural affections developing in the course of acute exanthemata. *Arch. f. Ohrenheilk.*, vol. xvii., p. 16, etc. Among 1,193 aural patients treated by Gottstein in three years, 66 cases, or 5.5 per cent. were due to scarlatina, measles, and variola. G. describes here fully a case of acute desquamative inflammation of the drum-membrane in the course of measles, combined with perforative, purulent inflammation of the middle ear, recovery; and an additional case of throat diphtheria and *otitis diphtheritica* sinistra in the *second week of measles*, also with recovery.

4. L. Blau. On aural affections occurring in acute infectious diseases. *Deutsche med. Wochenschr.*, 1881, No. 3. According to Blau, aural diseases, following measles, principally the purulent ones, may take a very severe course.

5. L. Blau. Diphtheritic inflammation of the external meatus after morbilli, followed after eleven days by diphtheritis faucium. *Berliner klin. Wochenschr.*, 1884, vol. xxi., p. 33.

6. Contributions to the statistics of aural diseases, by Dr. K. Bürkner. *Arch. f. Ohrenheilk.*, 1884, vol. xx., p. 81. We read on page 85: "Measles seem more seldom to cause aural affections. Of all patients, 3.0 per cent. ascribed their aural affections to morbilli, according to Kramer; 2.3 per cent. according to Schmalz; 2.7 per cent. according to Harrison; 1.5 per cent. according to Wilde; 0.1 resp. 0.4 according to Zaufal, and 3.2 per cent. according to *my* notes (epidemic of measles in Göttingen, winter 1881–1882)."

7. A. Tobeitz. Morbilli: clinical and pathologico-histological studies. *Arch. f. Kinderheilk.*, vol. viii., p. 321. Otitis media was the most frequent complication, 16 times among 73 convalescents. From the anatomical examinations of the hearing organ of the fatal cases there results, with all probability, a greater frequency of diseases of the ear. Of 22 children who died, the tympanic cavity was found, on examination, diseased in 17 cases. redness and swelling of the mucous membrane, destruction to the bone, obstruction with muco-purulent or putrid masses,

Clinical symptoms of disease were manifest in only 7 among these 17 cases. Tobeitz found in two, deceased on the first and third days, resp. after the appearance of the exanthema of measles, this otitis bilateralis also at the autopsy. Tobeitz supposes, therefore, that the middle ear, as well as the conjunctiva and the respiratory organs are *independently* affected in measles, but the affection may, clinically, not become evident before the stage of inflammation. The exudation of the, always lobular, pneumonia of measles is cellular, and tending to necrosis. The micrococci discovered in this affection are not concerned in the process of measles as such.

Literature Concerning Measles as an Etiological Factor of Acquired Deaf-Mutism.

1. "Diseases of the Ear," by I. M. Itard, German translation, 1822, p. 445, chapter xviii. On deafness by metastasis. Itard considers measles the disease which most frequently destroys the hearing by metastasis.

2. F. L. Meissner, "Deaf-Mutism and Instruction of Deaf-Mutes," Leipzig and Heidelberg, 1856, says, on page 158: "Extensive experience leads us to agree with Itard."

3. "Extension of Blindness, Deaf-Mutism, Idiocy, and Insanity in Bavaria," etc., by Dr. G. Mayr. No. 35 of the contributions to the statistics of the Kingdom of Bavaria, Munich, 1877. No. 42: In the year 1840 there were in Bavaria, among 2,897 deaf-mutes, 22, and in the year 1858, 3, in consequence of *variola and measles.*

4. A. Hartmann, "Deaf-mutism and Instruction of Deaf-Mutes," Stuttgart, 1880, mentions in table xii., p. 76, among 832 cases of acquired deaf-mutism, 30 in consequence of measles.

5. According to A. Hedinger, "The Deaf-Mutes and the Institutions for Deaf-Mutes in Württemberg and Baden," there was among 207 cases of acquired deaf-mutism in the two institutions of Baden, one case, or 0.48 per cent. after measles.

6. Schwabach. Deaf-mutism. S. Eulenburg's "Real-Encyclopædie." He mentions the rare occurrence of measles as cause of acquired deaf-mutism.

7. H. Schwartze, "Diseases of the Ear," Stuttgart, 1885, mentions, on p. 368, "larger blood extravasations into the labyrinthian cavity and into the membranous labyrinth," also in measles.

The statistics collected from the above-mentioned literature, except those of Tobeitz, indicate a comparative rareness of aural diseases in general after measles and of severe labyrinthian affections in particular. These statistical results offer a kind of consolation for the severity of the changes produced by the virus of measles in the labyrinth.

Explanation of the Illustrations.

Fig. 1 (Plates I. and II.).

Part of a radial section through the first cochlear turn and the neighboring region of the modiolus of the left petrous bone. That portion of the section is represented which lies nearest to the cochlear root. Hartnack, *22*, tubus o.

We see in the modiolus portion, as far as possible to the left and below, two tortuous, dull and thickened fragments of the arter. auditor. intern. = g, partly limited by an upward concave line, an indication of a perineurium, which had become structureless = s p. Further upward a rust-colored hemorrhagic exudation in process of pigment metamorphosis.

g sp = ganglion spirale.

s t = scala tympani.

s v = scala vestibuli.

l sp = ligamentum spirale.

st v = stria vascularis.

v d c = stunted ductus cochlearis.

p = a bright, periosteal stratum of delicate fibre on the medial wall of the scala tympani with a vascular defect ; p′ = the same with two thrombosed vessels.

Both scalæ are filled, the scala tympani completely, the scala vestibuli partly, by a mass consisting mostly of lymphoid cells in osseous transition, the smallest portion of broken-down lymphoid cells without nuclei ; this area appears bright in specimens stained with picrocarmine, the remainder, which is in osseous transition, and which is represented by the portion designated by $\frac{3}{6}$ in fig. 2 (plates I. and II.) appears red.

The ossified masses are in contact with the periosteum of the cavities in question, but not blended with them. It is clear that a proliferation of the periosteum must be excluded. It is easy to discover that a filling of the scalæ was gradually brought about by a *stratiform apposition* of the cell masses. The sulcus spiralis

internus is filled by a mass, not morphologically discernible, and forms a connected and compressed whole with the papilla acustica, the membrana tectoria, and the region of Claudius' cells.

Fig. 3 (*Plates I. and II.*).

Transverse section through the membranous and osseous semicircular canal and the neighboring bone. Hartnack $\frac{3}{5}$, tubus o.

K = bone.

E = endolymphatic.

P = perilymphatic cavity.

h-h = membranous semicircular canal.

p = periosteum.

g = thrombosed vessels.

g^1 = thrombosed vessel with hyaline thickening of the wall.

z = group of cells with one and more nuclei.

P^1 = perilymphatic cavity with hyaline degeneration of reticular structure.

The outline of the osseous and membranous semicircular canal, which in the normal state is sharply defined as well as their elliptical form, is lost. The structure of the osseous frame of the semicircular canal is indistinct in parts, the periosteum partly unravelled, partly atrophic (*cp.* the periosteal lining at P^1). The endolymphatic cavity is almost entirely filled, chiefly by a homogeneous mass of detritus, with which (on the right of the drawing) a number of spherical hyaline structures are connected.

The epithelial layer of the membranous canal is lost; it is entirely lacking; the propria (in the upper half of the drawing) may only be in part plainly distinguished. The connective-tissue layer, the membranous bands and their endothelial cells, form with each other and with the mostly swollen periosteum, a coherent structure of a partly reticular, partly cactus- and grape-like hyaline mass. The blood-vessels are thrombosed at g^1 with marked hyaline thickening of the wall. At z a group of mono- and poly-nucleated cells is seen. The region indicated by two lines $<$ is represented in the following figure with higher power.

Fig. 4 (*Plates I. and II.*).

Hartnack $\frac{3}{8}$, tubus o.

In this enlarged representation the grape-like form of the hyaline degeneration is plainer, the condition of the hyaline masses, which are again partly molecularly disintegrated, more coarsely

granular on the left and below the blood-vessel, which is traversed by the section, and whose wall is considerably thickened, more delicately granular below the longitudinal section of the blood-vessel.

In the latter is specially noteworthy (1) the condition of the *wall.* Its bright appearance is interrupted by dark long and short lines, apparently still the indications of the cellular elements in hyaline transition. And (2) the contents. The blood-corpuscles are irregular and mostly polygonal in consequence of the formerly existing globar stasis.

Fig. 5 (*Plates I. and II.*).

Part of a *vertical section* through the right cochlear capsule. The drawing is taken from a spot situated 1 *mm* medially from the mucous membrane of the promontory. Hartnack $\frac{3}{5}$, tubus o. Specimen stained with picrocarmine.

The specimens represent the initial and final stage of the change in the bone due to obliteration of the Haversian canals. The initial stage is represented in the lower $\frac{1}{3}$ of the drawing. The irregular appearance of the bright spots in the lower $\frac{1}{3}$ is caused by the basis-substance in the act of decalcification, which remained unstained by picrocarmine. The osseous areas lined by them, in which the bone corpuscles have partly lost their processes, were stained pale-red with picrocarmine. The upper $\frac{2}{3}$ represent, as far as they are characterized in the centre by large lacunæ, the final stage of the bony change due to obliteration of the Haversian canals. One part of the basis-substance of bone is still preserved in form of bridges and strips, which are covered in part by products of disintegration ; another abounds in the shape of arch-like lines, which are connected with each other by small and large bright spaces.

These are complete osseous lacunæ, originating from the breaking down of the osseous tissue into detritus, with subsequent absorption. The Haversian canals (on the left of the illustration) contain only thready fibrin, and present a streaked appearance ; another canal (on the lower right) shows shrinking of the contents besides the striated appearance.

The osseous tissue, situated on the right and left from the above-described upper $\frac{2}{3}$, shows pale-red coloration, finely fibred appearance, partly with stunted and partly without any bone corpuscles ; the same holds good for the immediate neigh-

borhood of the obliterated Haversian canals. The Haversian canals on the left remained colorless, but every thing else showed yellow picrin staining, as represented in the illustration.

Fig. 6 (Plates I. and II.).

Part of a *horizontal section* through the right cochlear capsule. The drawing is taken from a place in the bone, which is situated 2 *mm* laterally from the inner cavity of the second cochlear turn. Hartnack $\frac{3}{7}$, tubus No. 0. Glycerine specimen.

The changes represented in the drawing are, on the whole, the same as those presented above as the final stage of the upper $\frac{1}{3}$ in fig. 5, on the longitudinal section, only differing in their being extended over a larger territory. Besides the numerous interspaces due to the destruction of the bone tissue, there is seen in the centre of the drawing a still preserved oval island of bone, with bone corpuscles without processes, surrounded partly by decalcified basis-substance, partly (on the left and below the bone-island) by an interspace. Both Haversian canals (on the right of the illustration) are thrombosed. The preserved osseous stroma is chiefly covered by a finely grained, homogeneous detritus, or the meshes produced by it enclose these fine masses of detritus.

Fig. 7 (Plates III. and IV.).

From a transverse section of the capitulum stapedis of the right temporal bone. Hartnack $\frac{3}{8}$, tube drawn out.

H K = Haversian canals.

bk and bk′ = remnants of parietal blood corpuscles.

As far as H K extends the calibre of the Haversian canals is unchanged ; in consequence of the stasis, the blood-corpuscles are mostly polygonal. The greatest part of the Haversian canals is considerably enlarged by partly roundish, partly oval colloid bodies. Remnants of blood-corpuscles are seen only at isolated points of the space, whose walls are enlarged and from which the epithelium is absent. The epithelium also is wanting throughout the whole length of the lower wall, between bk and bk′.

Fig. 8 (Plates III. and IV.).

From a radial section through the second cochlear turn and the adjoining cochlear capsule. Hartnack $\frac{3}{8}$, tubus 0.

1. Osseous cochlear capsule. 2. External or periosteal layer of the ligam. spirale. 3. Region of the middle or loose layer

(atrophied). 4. Inner layer, g = thrombosed vessels. 5. Transsparent substance of the crista basilaris, which, at 6, goes almost at right angles into the membr. basilaris instead of a concave curvature, as in the normal state, while the mb. is curved instead of horizontal. Claudius' and Boettcher's cells are missing in this region. In their place products of disintegration are seen. Between 5 and 6 the cells of the sulcus spiralis externus are also wanting. Instead of these mono- and poly-nucleated cells are seen. 8. Region of the stria vascularis. 9. Reisner's membrane. 9′. Its insertion to the detached ligam. spirale. 9″. Reisner's membrane atrophied at its insertion. 10. The radial and medullated nerve-fibres between the layers of the lamina spiralis ossea. The numerous regularly recurring light spaces are due to lost nerve-fibres. Cr. = Crista spiralis. 11. Its labium vestibulare is infiltrated with lymphoid cells. This infiltration continues to 9″ and 15, beyond the margin of the crista spiralis. 12. Labium tympanicum of the crista. This presents chiefly a homogeneous appearance, in smaller portions a striated one, except at its inner portion, where (to the right of 9″) on the upper surface of the nerve extension a few spindle-like, radially arranged nuclei of the periosteal layer of the crista may be seen. 13. Membr. Corti, or tectoria, is visible *en face* and dislocated. The tectoria is chiefly blended with Reisner's membrane ; the blended portions are concavo-convex. 14. Marginal cord of the tectoria covered by mono- and poly-nucleated cells and products of detritus. 15. Its inner fixed insertion in the act of disintegration. 16. Sulcus spiralis internus ; its epithelium partly in the act of disintegration, partly wanting. The transparent substance of the labium tympanicum of the crista (12) in the act of disintegration, narrowed, and its surface furrowed, instead of being smooth ; under the surface products of disintegration, partially covering the initial portion of the basilaris, hence its transition into the latter is indistinct. The nucleated layer of the basilaris devoid of nuclei, the initial portion of the latter is striated. The tympanal lining stratum (*tb.*) of the basilaris apparently widened. This is due to an infiltration of non-nuclear as well as mono- and poly-nucleated cells, which extends into the area of the nerve on its lower surface (near 12). 17. Inner, 18. Outer rods of Corti's arches. 19. Outer supporting cells.

With regard to the characteristic change of the ligamentum spirale, we are best instructed by the large crescentic, almost

entirely empty space (3), in the lower portion of which a large colloid body is situated, which has been forced arch-like into the corresponding portion of the ligamentum spirale (in the normal state it is concave towards the scala vestibuli). The falciform space represents the so-called *middle loosened layer* (3) of the ligamentum spirale (Schwalbe) perished by tissue disintegration, whose ultimate product is the hyaline body. The *outer*, in the normal state, dense, periosteal layer (3) is preserved, but loosened and has mostly lost its former abundance of nuclei. The adjoining bone is also changed (*cp.*, the text). The right lower five oval spaces are veins with shrunken contents. The left thrombosed vessel, situated to the left and somewhat below the colloid body, is a vein. Downward and inward from this we see an atrophic portion, which, as well as the thrombosed vein, *belongs to the inner and, normally, dense layer* (4) *of the ligamentum spirale.* This third wide layer of the ligamentum spirale, which normally consists of densified connective tissue, abundant in cells, has a brighter appearance than in the normal condition, on account of one portion of the still preserved cells being devoid of nuclei, another being entirely destroyed. The extreme stage of atrophy is found below the colloid body, upon its inner and lower portion (*cp.* the text above).

Upon the inner surface of the inner layer of the ligamentum spirale, facing the sulcus spiralis externus, upward from the insertion of the basilar membrane, we see the translucent limiting membrane (5) in connection with the basilaris, forming its modified continuation. It is studded with a number of mono- and poly-nucleated lymphoid cells, which have partly lost their nuclei. Their epithelium is wanting ; it is lost. Where it ends the considerably changed stria vascularis (5) begins. This, in the normal state intimately connected with the inner layer of the ligamentum spirale, has become detached from it by the pathological process ; the point of detachment is indicated by an arch-like interspace, expanding upward to the insertion of Reisner's membrane (g'). Reisner's membrane is hereby already lowered at its insertion and its angle of insertion, outer-upper or vestibular angle, becomes obtuse, while in the normal state this does not occur below the third turn. Partly in consequence of this detachment, partly through the morphological change, the shape of the ligamentum spirale in the entire region of the scala vestibuli is changed beyond recognition. In the normal state[1] concave from the

[1] *Cp.* Fig. 1.

insertion of Reisner's membrane to the prominentia spiralis, on this place convex, further downward again concave, and throughout with a sharp edge, the big difference is well marked, notably by the absence of the prominentia spiralis and its vessel. The blood-vessels of the ligament are obliterated. Instead of the ligament we see an irregular layer of mono- and poly-nucleated cells, which are devoid of nuclei.

Corti's organ is considerably changed. Upon the membrana reticularis, to the end of the outer supporting cells, we see, partly, a number of hyaline bodies whose walls are already in the act of granular disintegration, partly fine detritus and coagulated lymph. The inner supporting (the hair) and the epithelial cells, situated inward and below these, are wanting. Instead of these we see detritus, a few mono- and poly-nucleated cells, the largest ones being outward from the body of the inner rod.

The nerve-fibres passing in- and outward from the inner rod of Corti's arch to Corti's organ, are absent. The configuration of Corti's tunnel (between 17 and 18) is irregular, in consequence of the altered curvature of the arch, especially the inner one. The basal cell of the outer rod, as well as the protoplasmatic layer, are wanting.

Specially noteworthy is the change in the region of the outer supporting cells. The membrana reticularis extends only to the fourth last outer supporting cell. The second last is already atrophied ; its dislodged nucleus lies next to the third last. The limiting walls also of the the four last supporting cells are already in the act of atrophy, and their fine products of disintegration are confluent with those of Claudius' cells. The basal portions of Deiters' cells are partly in hyaline degeneration, partly atrophied. Of their phalangeal processes the process of the second and fourth cells is the most plainly visible. The atrophy of the hair-cells is further advanced than that of Deiters' cells. We see the remnants of the first and fourth hair-cell adhering as hyaline bodies to the membrana reticularis.

Fig. 9 (*Plates V. and VI.*).

From a radial section of the second cochlear turn of the right petrous bone. Hartnack $\frac{3}{8}$, tubus drawn out.

1. Radial medullary nerve-fibres. 2. Crista spiralis. Its surface, instead of being slightly convex, is irregularly convexo-concave, studded with mono- and poly-nucleated cells. This in-

filtration continues into the sulcus spiralis internus. The transparent layer, the vitreus of the crista, is dull. The processes of the stellate bodies of the propria are absent. The propria and the periosteal layer form a stratum, interlaced by radially arranged nuclei, upon whose surface up to the neighborhood of the labium vestibulare (3) lies a cylindrical epithelium, the outlines of which upon the inner half of the row of cells are irregular and indistinct on account of beginning disintegration. 3. Its labium vestibulare is stunted (*cp.* text). 4. Its labium tympanicum, between both the sulcus spiralis internus, is flattened, instead of being concave. 5. Reisner's membrane. 6. Transition of the labium tympanicum into the basilar membrane. 7. Nuclear layer of the basilar membrane. 8. The so-called tympanal lining stratum infiltrated by mono- and poly-nucleated cells. 9. Epithelium of the sulcus spiralis internus, partly extremely granular. 10. Corti's membrane, with its marginal cord. 11. Inner; 12. Outer Corti's rod. 13. Outer supporting cells. 14. Remnants of atrophied Corti's cells. 15. Atrophied basal pieces of Deiters' cells, compressed by a transversely oval, hyaline structure. 16. The epithelium of Reisner's membrane (5) is wanting; it is mostly covered by products of retrogressive metamorphosis; between its inner portion, which is here abnormally convexo-concave, and the centre of Corti's membrane, or m. tectoria (10), coagulated lymph is found; the tectoria is detached from Corti's organ and placed upon its surface. *All structures of Corti's organ, situated in front of the inner Corti's rods, are completely destroyed, viz., the inner supporting cells, also the nerves running to the inner auditory cells, the inner auditory cells themselves, and the epithelial cells* (*Retzius*), *situated inward and below the auditory cells.* Instead of these we see between 4 and 11 a group of polynucleated cells, lying outward and somewhat upward from 11 upon the inner arch, two oval, hyaline structures (possibly products of degeneration of the inner hair cells), the latter surrounded partly by some polynucleated cells, partly by coagulated lymph, which covers and encloses the entire remaining Corti's organ.

Of both Corti's arches, only the outer one shows fibrillar structure, the inner only upon the upper portion, the basal portion being homogeneous. The outer basilar portion is lacking. We see Corti's tunnel, filled by an oval structure, and enclosed by an anchor-like, dark, homogeneous mass, partly contiguous to the inner rod, and by a bright, hyaline mass, which embraces the

inner surface of the outer rods. Of the phalangeal processes of Deiters' cells, we see only two distinctly, viz., the first and the last, but all their basal portions, stunted, and approximated by a large, transverse, oval structure, situated between the last Deiters' and the first outer supporting cell.

We see three stunted hair-cells without hairs, two of which, the two first, have distinct processes directed towards the basilaris.

The structure of the outer supporting cells still appears normal, except the indented condition of their boundaries, directed toward the ductus cochlearis, which in the normal state is convex.

Fig. 10 (*Plates V. and VI.*).

An additional radial section from the second cochlear turn of the right petrous bone. Hartnack $\frac{3}{5}$, tubus drawn out.

1. Radial medullary nerve-fibres at 1′ disintegrated. 2. Crista spiralis, almost entirely covered, *i. e.*, the inner half by nucleated, and its outer half by cells without nuclei, and by products of disintegration. 3. Its stunted labiumve stibulare. 4. Its labium tympanicum. The transition into the basilar membrane hidden by overlying products of disintegration of the nerve and invisible. The surface of the labium tympanicum is partially disintegrated, thereby presenting a fimbriated appearance. 5. Nuclear layer of the basilaris partially covered by products of disintegration. 6. Basilaris propria, only its outer half visible. 7. The so-called tympanal lining stratum infiltrated by mono- and poly-nucleated cells. The infiltration partly extends into the membrana basilaris, which, therefore, appears indistinct. 8. Corti's membrane with its marginal cord. 9. The flattened sulcus spiralis internus with its epithelium, upon which products of disintegration are situated. 10. Region of the inner rod. 11. Outer rod. 12. Outer supporting cells. 13. Disintegrated nerve running to the inner portion of Corti's organ. The structures of Corti's organ, situated in front of the inner rod, are entirely missing, as in fig. 9. Instead of the inner rod (10) we see a pear-shaped hyaline structure partly covered by an oval, dark, nucleated cell, the nucleus itself being highly granular. The body of the inner rod is missing; only its head-piece is seen. In the corresponding interspace we see a large and next to it a smaller hyaline structure, both surrounded below by an isolated cell, above by a group of polynucleated cells, the entire territory in question being outwardly limited by molecular detritus.

The outer rod (11) has lost its delicate, dark, longitudinal striæ, partly upon the basilar portion, its head-piece, as well as that of the inner rod, being more striated than in the normal state. The protoplasmatic lining and the basilar cells of both rods are absent. Instead of these we see Corti's tunnel chiefly filled by a round and hat-like hyaline structure, the latter with two nuclei. The cell upon the outer rod (11) is perhaps the outer basilar cell, which has advanced further upward. The entire condition probably represents a product of metamorphosis of the basilar cells and of the protoplasmatic lining of the rods. The group of cells between 4 and 13 is a remnant of the perished portion of Corti's organ, situated in front of the inner rod. Nerves are not seen to enter Corti's tunnel. Three stunted hair-cells (14) are seen, all without processes, and just as many changed Deiters' cells.

Of the Deiters' cells (15) the first is blended with the first hair-cell, both are stunted, the last, the fourth Deiters' cell being the least stunted. It represents a concavo-convex arch, apparently interrupted by an overlying cellular structure, which can be traced to the neighborhood of the basis. The basal portion of the first Deiters' cell is in hyaline degeneration, between which and the basal end of the last an irregular dull granular mass is situated. The Claudius' and Boettcher's cell are absent. The entire Corti's organ is enclosed partly by coagulated lymph, partly by molecular detritus.

Fig. 11 (*Plates V. and VI.*).

Radial sections through the second cochlear turn of the *left* petrous bone. Hartnack $\frac{3}{4}$, tubus O.

1. Bone. 2. Inner or periosteal stratum of the lig. spirale, loosened at $2'$, inwardly a hyaline band; downward from 2 at $2''$ we still see the nuclei of the periosteal layer, in the remaining portions to the parts below more or less large defects, due to tissue disintegration. This is also true of the middle and inner layers in the upper portion, and of the middle layer in the lower portion of the drawing. g = thrombosed vessels. 3. Transparent substance, continuation of the propria of the basilaris. 4. Region of the prominentia spiralis in the act of atrophy and disintegration with a thrombosed vessel, downward a group of lymphoid cells, which further downward are lying upon the transparent substance (3) of the basilaris. In the direction of the outer supporting cells the basilaris shows indications of the disintegrated Claudius'

cells. 5. The tympanal lining stratum, infiltrated by lymphoid cells, up to the radial medullary nerve-fibres. The layer which lies nearer the labium tympanicum is brighter, and in the act of atrophy. 6. Radial medullary nerve-fibres, their entrance into the epithelium (*a*) atrophic; beyond their entrance, granular products of disintegration. 7. Labium tympanicum of the crista spiralis. 8. Its transition into the basilaris. 9. Region of the sulcus spiralis internus; the inwardly situated epithelial cells partly spherical, in beginning disintegration, instead of low, as in the outward portion. 10. Region of the inner supporting and hair-cells, which are lacking. In their place upward some detritus. 11. Inner Corti's arch defective. 12. Outer Corti's arch. Its body proper separated from the head and basilar portion, and somewhat dislocated with the separated ends. Basilar cells and protoplasmatic layer of both rods absent. 13. Nuclear layer of the basilaris, covered by products of disintegration; these products partly cover the adjoining propria of the basilaris. 14. Outer supporting cells, their outer wall partly covered by polynucleated cells. The protoplasma of the four outer supporting cells is homogeneous, and brighter than that of the remaining portion. The greatest portion of Corti's and Deiters' cells is atrophied. Of Deiters' cells, the last (*d*) is preserved best. *We emphasize that the process of one Corti's cell*, the cellular body of which is in hyaline degeneration, *is still preserved running towards the basilaris.* The inner portion of the membrana reticularis is absent.

Explanation of the Colored Plate (*Tab. VII.*).

Fig. A.—From the perilymphatic space of the sagittal membranous semicircular canal of the left petrous bone. From a visual field of the hyaline degeneration, with partial disintegration of the hyaline masses. *Hartnack.* Oil immersion $\frac{8}{12}$. Tubus 0, 650:1. We see chiefly mono- and strepto-cocci, now and then also irregularly arranged groups and clusters of cocci.

Fig. B.—From a medullary cavity of the left petrous bone. *Hartnack.* Oil immersion $\frac{4}{12}$. Tubus 0, 1000:1. We see chiefly mono- and diplo-cocci, isolated chains, and clusters of cocci. The medullary substance was partially destroyed, therefore a portion of the medullary cavity empty.

Fig. C.—From a Haversian canal of the left petrous bone. *Hartnack.* Oil immersion $\frac{4}{12}$. Tubus 0, 1000:1. We see mono- and isolated tetra-cocci; but the chain-form is, upon the whole, prevalent.

Fig. D.—From a Haversian canal of the right petrous bone. *Hartnack.* Oil immersion $\frac{4}{12}$. Tubus 0, 1000:1. All forms mentioned before are also represented, but in smaller number.

REVIEWS.

The Surgical Treatment of Brain Diseases. Prof. E. von Bergmann, Berlin.

The range of operative surgery grows ever wider. A technique steadily improving, conjoined with rigorous antisepsis, leads continually to fresh triumphs. A successful nephrectomy is no longer a rarity.[1] Langenbuch[2] has done a partial extirpation of the liver with good results, and Victor Horsley has led the van in the surgical treatment of tumors of the spinal medulla, with the brilliant removal of a myxoma which had, for three years, occasioned the most severe symptoms.

The surgical treatment of brain diseases has a longer history, thanks not only to the advances in surgery proper, but also to the untiring researches of the physiologists and brain-pathologists, in connection with the localization of function.

Among the affections which are accessible to surgical interference, and which have been repeatedly operated with success, belong cerebral abscesses due to otitis. The first case of this kind operated and cured was by Schede, and was reported by Truckenbrod in these Archives, vol. xv., p. 170.

At the international otological congress held at Brussels in 1888, Thomas Barr reported six such cases, with a permanent favorable result in five. Von Bergmann,[3] in his model essay on brain surgery, had already, in the early part of 1887, developed the subject of the operative treatment of these affections.

It would be a grave sin of omission were we to content our-

[1] Schede has obtained eleven cures in twenty cases operated by him. *Deutsche med. Wochenschr.*, 1888, No. 52.

[2] *Berliner klin. Wochenschr.*, 1888, No. 3.

[3] Arbeiten aus der chirurgischen Klinik der Königl. Universität Berlin. Herausgegeben von E. v. Bergmann. V. Die chirurgische Behandlung von Hirnkrankheiten, S. 50–164.

selves here with a passing reference to this most important work, especially as far as it deals with the treatment of ear diseases. A somewhat extensive review of it, however, will not render it less necessary for my colleagues to make a careful study of the work itself; to read it merely were not enough. It may happen to any aurist, even though his practice be limited, to have the question presented to him whether the aid of a competent surgeon may not help him to save the life of a patient. The study of the essay and the review will be of considerable assistance in deciding the important points presented in such a case.

In many cases, indeed, his conscience will remain troubled until his surgical colleague has answered "Yes" or "No" to the question of the advisability of operative interference. Bergmann speaks thus: "It is just in this district of brain surgery that it has happened to me more than once that very able colleagues have invited me to perform operations which I felt obliged to refuse. Such differences have suggested to me the advisability of bringing about a better understanding between physician and surgeon by a presentation of the attitude which surgery, according to my view, must take with regard to a certain class of brain diseases. One can well understand how, in these considerations, I think primarily of the interest of the surgeon. My personal tendency, or, rather, my conviction, inclines me to narrow rather than extend the range of surgery. Nowadays we are apt to dare every thing without reflecting as to the aim and reward of the venture. I think that I am not limiting too much the proper field of surgery if, in the accompanying clinical studies, I first investigate the conditions under which surgical interference has good, or I will even say the best, chances of success, and then recommend operation for such cases, and such only."

My colleagues who have had experience with abscesses of the brain, following ear disease, and are familiar with their clinical signs, course, and pathological appearances, will agree with me that the principle laid down by our eminent surgeon is the only reliable one. One needs but to recall the cases he has seen run a typical course, and contrast them with those others in which post-mortem examination reveals multiple abscesses; or, again, those in which several possible etiological factors are presented in connection with a doubtful disease-picture.[1]

[1] For example, in one case to which I was called, traumatism, syphilis, and a recently cured otorrhœa, entered into the question of the etiology of the disease. —*Reviewer.*

Deep abscess of the brain. As suppuration of the brain can terminate only in rupture into the ventricles, or the equally fatal diffuse meningitis, the opening of an abscess of the brain must be regarded as a means of relief from a most threatening danger to life. As to doubting the necessity of trepanning for brain abscess on account of the possibility of its encapsulation (lately Rose), or because of the attending difficulties of diagnosis, the author says: "The range of the first is overestimated, the difficulties of the diagnosis are gradually being overcome."

Only such brain abscesses as develop rapidly are found without a capsule, *i. e.*, a sclerotic thickening of the surrounding tissues. *Brain abscesses which have developed gradually and slowly are almost all encapsulated.*[1] This capsule does not represent the limit of the suppurative process, and does not protect the brain tissue from extension of the process. Post-mortem examination often shows breaks in the capsule; we can not regard the process, therefore, as an efficient limitation leading to a sort of cure in the way of retrograde changes. The hope that a collection of pus in the brain, if left to itself, may spontaneously find an exit through the ethmoid plate into the nasal passages, or through the roof of the tympanum into the ear, and, that so, a favorable termination may ensue, is not to be entertained. A fortunate issue can be expected only from the use of the trephine, to which the difficulties of diagnosis offer no longer the same insuperable objections, as heretofore.

In this connection the etiology is the main factor. Brain abscesses do not occur idopathically. They arise either from a traumatism or from a suppurative process in the skull, most frequently an otorrhœa. There are, however, metastatic and tuberculous cerebral abscesses; the former are merely incidental to pyæmia, and are therefore multiple. Exceptionally we find them single; they are so much easier to diagnosticate, as they are connected with the presence of pus collections in other organs.

From the opening of tubercular abscesses of the brain little is

[1] The question as to the shortest time in which encapsulation may take place, a question very important also in its medico-legal bearings, is, unfortunately, not yet settled. In a case of left-sided chronic suppuration of the middle ear, to which I was called a few hours before the fatal termination, the patient had been confined to bed only eight days, with severe pains in the ear and head. Symptoms of earlier cerebral trouble were denied both by the relatives and the two physicians in attendance. Autopsy: A carious spot, of the size of a pin-head, in the roof of the tympanum over the malleo-incudal articulation, basilar meningitis, and *two abscesses already encapsulated*, one in the left half of the cerebrum, the other in the right half of the cerebellum.—*Reviewer.*

to be expected. *A favorable issue is to be looked for only in cases caused by traumatism, or by suppuration of the ear or other bony parts. An early diagnosis in such cases is therefore of importance.* We must pass by without special notice that part of the work devoted to abscesses arising from traumatism. Our main interest is naturally centred in those connected with otitis. They constitute probably one half of all abscesses of the brain; this lacks, as yet, statistical proof. The suppurative process is always chronic (no brain abscess has ever been found connected with acute purulent otitis); its seat is the middle ear. It is generally the product of an otitis media chronica purulenta originating in youth.

The abscesses are seldom found immediately above the diseased tympanum; indeed, the greater number are separated by a layer of normal brain substance from the site of the primary suppuration.

"*The position of the deep abscesses under consideration is quite definite. With few exceptions, they are found either in the temporal lobe or in a cerebellar hemisphere.* The former situation is more common than the latter."

The author cites the following statistics of Barr. Of 76 cases of suppuration of the ear (7 of them coming under Barr's personal observation), the abscess was situated in 55 in the temporal lobe, in 13 in the cerebellum, in 2 in the pons, and in 1 in the crus cerebri. No statements are made with regard to the encapsulation of these abscesses, nor of the presence of sinus-thrombosis or meningitis as complications.[1] In 69 cases the pus was fetid.

[1] The Reviewer takes the liberty of inserting the following statistics of Körner, collected, indeed, with an entirely different object in view, that of proving and explaining the greater prevalence on the left side of diseases originating in middle-ear affections and caries of the petrous bone. Sixty-seven abscesses of the brain arising from otitis are divided into 5 groups. There are 31 abscesses of the cerebrum, 19 of the cerebellum, 4 of cerebrum and cerebellum, 8 of cerebrum with phlebo-thrombosis of the transverse sinus, 5 of cerebellum with phlebo-thrombosis of the lateral sinus. In 14 of the cases of cerebral abscess the temporal lobe was exclusively affected; with the temporal lobe affected there was involvement of the frontal lobe in 1 case, of the posterior corner of the opposite side in 1 case, of the occipital lobe in 1 case, and a complicating meningitis in 8 cases, in 1 of which the abscess was double.

The corpus stratum and thalamus opticus were the seat of the disease once, the right hemisphere twice, the parietal lobe once, the base of the posterior lobe once, the medullary substance of the first upper frontal and of the anterior central convolution. A comparison of the statistics of Barr and Körner shows the following difference. According to Barr the abscess is located in the temporal lobe in 72.3 % of the cases; according to Körner, if we disregarded complications, in 37.3 %, but if we include only uncomplicated cases, and none others should be submitted to operation, only 20.9 %. It would carry us too far to submit the other tables to a like analysis. This single comparison suffices to show that the pure cases are rarer than would seem from Barr's statistics, and that great caution must be exercised on account of complications and the multiplicity of the abscesses. (*Vid. Arch. f. Ohrenh.*, vol. xxvii., p. 126–139.)

"Abscesses of the brain begin insidiously, and herein lies the difficulty of the diagnosis." The author denies[1] that slight concussions, or other injuries of the head occurring in the course of ear disease, can lead to the formation of brain abscesses, because, in the cases in which death has followed such traumatism, the interval was too short and the abscess too large; more rapid growth, rupture, etc., of the abscess under these conditions, have been demonstrated. "I have twice had occasion, in the practice of otologists, to observe the rapid development of the abscess from the latent condition to the final stage, after trepanation of the mastoid" (!).

Suppuration with and without ostitis in other parts of the skull play only a very unimportant part in the history of brain abscesses. Whatever be their origin, we may divide them, according to the symptoms they produce, into three groups:

1. Symptoms, directly dependent on the suppuration.

2. Symptoms of increased intracranial pressure and of disturbing displacements of the brain substance (showing partial resemblance to those of tumors of the brain).

3. Focal symptoms corresponding to the site of the abscess.

In the diagnosis of otitic brain abscess, we can place no reliance on the sudden accessions of fever (chills and rise of temperature), which in cases of the traumatic form correspond with the periodical development of the chronic abscess,[2] because the suppurative process in the ear may produce like symptoms, as in retention of pus, etc. No greater reliance can be placed on disturbances of the general health.

One of the earliest and almost constant symptoms dependent on suppuration of the brain is *the pain in the head, caused by the increased intra-cranial pressure, and regularly becoming more intense during the febrile exacerbations.* It is often a fixed pain; in otitic abscesses of the temporal lobes or cerebellum strictly localized, corresponding to the situation of the abscess, and increased by percussion of the painful spot.[3] The other pressure-signs are, the terminal stage not being considered, rarely so well developed as in brain tumors. In the latter they are rather constant, in abscess

[1] In opposition to Griesinger.—*Reviewer.*

[2] In a very interesting case of traumatic abscess, under the care of Bergmann the temperature was one degree higher on the corresponding side of the head than on the other. The thermometer of v. Anrep was employed in making the measurements.

[3] The pain is increased by all movements of the head, including such as are communicated to it by coughing, or even hawking.—*Reviewer.*

they vary more. Slowing of the pulse—even to 30,—loss of consciousness, coma, may all intermit in abscess, the intermissions naturally being deceptive; in tumor such intermissions are very rare. On the other hand, the choked disc is much more common in the latter.[1]

The focal symptoms are of considerable importance when the abscess is situated in the motor region, but of very little value when in the frontal, occipital, or temporal lobes. For as long as the suppuration is confined to the medullary layer and the gray substance is preserved, the focal symptoms may be absent, even though the entire half of the cerebrum be involved; the more the abscess invadés the cortex, the more definite do they become.

In considering the advantages to be obtained from the evacuation of a brain abscess, it is of considerable prognostic value that the focal symptoms dependent on destruction of the brain substance will persist after the operation, whilst the circulatory disturbances in the neighborhood of the abscess and the symptoms resulting therefrom admit of recovery.

The author does not enter upon the discussion of the special focal symptoms and their important combinations. He holds that when the abscess is situated in the frontal lobe, it affords no clue for a diagnosis, as the entire lobe may be destroyed without special sign. "The nearer the abscess approaches the posterior division of the frontal convolution, the more apt are we to observe strabismus, disturbances of speech, and irritation or paralysis of the facial. Abscesses of the occipital and temporal lobes are characterized by the same absence of focal symptoms. Hemianopia, the characteristic focal symptom of the occipital lobe, was noted in a single case of abscess situated in this part. If ever, in a case of unilateral suppuration of the ear, deafness should develop in the opposite side without any peripheral disturbance of the hearing, and if at the same time there are symptoms which point to the existence of an abscess in the temporal lobe of the side first affected, the newly developing deafness may be regarded as a decisive focal symptom, as the physiologists locate the centre of hearing for each ear in the opposite temporal lobe.

The author reports only successful cases from his own practice

[1] We learn, however, from the reports of the cases given, that in large abscesses where the pressure on the cranial contents is considerable, the choked disc is present, and may be a very useful diagnostic symptom. The greatest difference in pulse rate has been observed, by the Reviewer, in a case examined immediately before and after rupture of the abscess into the ventricles. The pulse fell suddenly from 132 to 36; death ensued in a few minutes.

and that of others during the past few years (in the *Deutsche med. Wochenschr.*, 1888, No. 50, he has reported an otitic brain-abscess successfully operated more recently); he discusses in this connection, among other things, the observations of Schede-Truckenbrod on aphasia as a focal symptom in affections of the temporal lobe, and claims for them a certain importance.[1]

Bergmann recommends, as the best spot for trepanation with the chisel, "a region somewhat in front of and above the posterior angle of the parietal bone." The middle temporal convolution may be reached without injury to any of the main branches of the middle meningeal, if we enter at a point found by producing a line passing through the lower edge of the orbit and the auditory meatus 4 *cm* behind the latter, and erecting here a perpendicular 4–5 *cm* long. Syringing of the abscess after opening is disapproved of. Simple evacuation of the pus is sufficient. To determine the location of the pus the exploring needle must replace the knife. *It is not a matter of moment, under all circumstances, to discover every abscess that may possibly exist in the hemispheres of cerebrum and cerebellum, but rather to make sure of opening that of which clinical experience has enabled us to recognize the presence.*

If otologists, in connection with competent surgeons, will but act on this principle, we will be justified in expecting, in the course of time, fairly favorable statistics on operated otitic brain-abscesses. However, disappointments will be in wait for even the most cautious and skilful surgeon, as, for example, in those cases of multiple abscesses in which one runs a typical course, the other or others remaining latent. It must always be borne in mind that the carriers of infection,[2] which we may now regard as the mediate cause of the abscess formation, *wander simultaneously into various districts of the brain*, in any of which they are liable to

[1] I was once called by two colleagues to see a patient, himself a physician, shortly before his death, in order to determine if the brain affection had any connection with the ear disease. The patient had been from youth subject to an occasional left-sided otorrhœa. After a period of excitement and sleeplessness lasting eight days, he suddenly developed an ataxic aphasia in the midst of his professional duties. There could be no doubt of the connection between the ear disease and the abscess of the temporal lobe. The aphasia may be intermittent in cases where the course of abscess of the temporal lobe is very protracted. Such a case is described in Toynbee's work. Intermittent aphasia occurs further as an indirect focal symptom in tubercular meningitis of the convexity. When it is accompanied with fixed pain in the temporal region, as I once observed it, and that, too, almost immediately following the healing of a left-side suppuration of the middle ear, it may easily lead to error in diagnosis.

[2] In two cases observed and studied by me I found bacilli and enormous masses of micrococci, but no streptococci in the abscess-membrane.

settle and develop. This wandering, according to Binswanger, occurs most probably through the lymphatic canals of the connective tissue, and explains the fact which pathologists and otologists have long since known and wondered at, that abscesses of the brain, as a rule, are separated by a more or less wide zone of healthy brain substance from the original foci of inflammation in the ear, and that further, a second collection of pus is not infrequently found in the opposite half of the brain. (*Reviewer.*)

TUMORS OF THE BRAIN.

"If a brain-tumor is to be operated, it is not enough to know the position of the growth; it must be ascertained also if the tumor admits of enucleation."

Basing his conclusions on the statistics of one hundred tumors of the brain, collected by White, Bergmann shows in how small a proportion of the cases the operation is indicated. The results in the cases thus far operated, in all eight, are accordingly poor. In five, death ensued as a result of the operation; in but one, Horsley's, has a permanent cure (reported four months later) been obtained. An exploratory trepanation is absolutely contra-indicated when a positive diagnosis of the locality of the tumor cannot be made from the focal symptoms. Hemorrhage and œdema of the brain are serious dangers which may result from the operative interference. (Compare the reports of the cases.)

EPILEPSY.

The traumatic epilepsy developing after head injuries is to be regarded as a cortical epilepsy after Jackson. In considering their treatment it is well to study their analogy with those produced experimentally by irritation of the parts of the brain-cortex presiding over motion. Horsley deserves also here the credit of leading the way. Two cases operated by him may be regarded as cured.[1] He holds that all the parts that look diseased must be removed. The operation for epilepsy is allowable and indicated "when we have full reason to believe in the presence of a palpable and removable lesion of the brain-cortex. This we have, however, only in such cases in which the convulsive movements begin, advance, and finally become generalized in exactly the same way as in the experimentally produced cases in the animal."

[1] Very recently, Keen, of Philadelphia, has operated successfully three cases of traumatic epilepsy. They proved to be tumors of traumatic origin. Comp. *Deutsche med. Ztg.*, 1888, No. 87.—*Reviewer.*

We have endeavored, so far as space permitted, to present to our readers the essence of this most valuable work. The nicer details and practical hints are to be found mainly in the clear and brilliant comments added to the elaborate case-reports, and to these we would again bespeak special attention.

S. Moos.

2. **The Ear and its Diseases** (being practical contributions to the study of otology). By SAMUEL SEXTON. Edited by C. J. Colles, M.D. New York: W. Wood & Co., 1888. Price, $4.00.

Sexton's well gotten-up book of 461 pages, is a semi-systematic treatise of otology, treating at exhaustive length subjects to which the author has devoted special attention, whereas others are very cursorily dealt with. In fact, the bulk of the book consists of the republication of papers, here and there somewhat changed, which the author has contributed to various periodicals during the last fifteen years. There is a good deal of suggestive original matter in the book, at the side of not a little of a dogmatic and speculative character, setting boldly the authority of the writer against the authority of all the world. In some subjects the mental idiosyncrasy of the author is apparent,—for instance, in the ubiquitous dental reflex and a leaning towards specific medication.

While reading the book we jotted down a number of remarks which we thought would interest our readers.

The improvement of hearing and relief from autophony and nervous symptoms which many patients experience in pulling the auricle in various directions, are explained by the change of tension of the drum-head by the exterior muscles and fascia of the ear. In an anatomical specimen prepared by the author and Dr. S. H. Pinkerton, alternate tension and relaxation of the fascia, tightened and relaxed the *Mt*, the motions being perceptible to the eye (pp. 21 and 22). Chapter III., "The Aëration of the Tympanum," contains many quotations from, and Chapter IV., "The Sense of Hearing," draws extensively on, a paper by Dr. W. Rutherford (published in *The Lancet*, 1887). Helmholtz's theory of sound-perception must be abandoned in favor of W. Rutherford's telephone theory of sound-perception which the author adopts. "This theory is that the cochlea does not act on the principle of sympathetic vibrations, but that the hair-cells vibrate to every tone just as the drum of the ear does; that there is no analysis of complex vibrations in the cochlea or else-

where . . . ; that the hair-cells transform sound-vibrations into nerve-vibrations which, arriving in the sensory cells of the brain, there do not produce sound again, but the sensations of sound, the nature of which depends not upon the stimulation of different sensory cells, but on the frequency, amplitude, and form of the vibrations coming into the cells, probably through all the fibres of the auditory nerve."—(pp. 56, and 57.) "Should my theory of the sense of hearing find acceptance, it will lead to a reconstitution of the theories regarding the other sense organs." This theory, by which it would be difficult to explain diplacusis and other phenomena, will be completely demolished when microscopic studies continue as they have begun to demonstrate that tone defects in the field of audition depend on the destruction of certain portions of the cochlea, as defects in the field of vision are shown to depend on the destruction of certain portions of the retina. There is no need, as yet, to reconsider the theories of the other sense organs.

In Chapter V., "The Common Causes of Ear Disease" are rather scantily treated. Some of the causes are not even mentioned,—for instance, the characteristic and most important deafness from hereditary syphilis, the occurrence of which, in conjunction with heredito-specific (parenchymatous) keratitis, is by no means rare. In Chapter VI., to make amends, the causes of catarrh of the upper air tract are discussed at great length, especially the meteorological influences.

The reflex influence of oral irritation is detailed in Chapter VII., and passes as a red thread through the whole work. To define his position in this respect, the author says: "That all morbid conditions of the mouth may exist without serious or recognizable aural affection cannot be denied (p. 97). . . . In his aural practice he has observed a great many recoveries quickly following the removal of defective teeth" (p. 96). Granting the correctness of the author's assurance, I expect reciprocity of belief on his part when I sum up my personal experience as follows: I have seen cases, a good many of them—some even from the author's practice—in which the removal of the incriminated teeth showed no influence whatever on the ear trouble.

Chapter VIII., Sea-bathing, etc. Nothing new.

Othæmatoma (Chapter IX.) is very extensively described, though the author, in conformity with earlier writers, makes no distinction between it and the simple—non-hemorrhagic and

non-traumatic—perichondritis. He recommends not to be too hasty in interfering, as a certain number of cases tend to spontaneous recovery. Pressure has proved unsatisfactory, in his experience. With proper discrimination he recommends massage, a silk seton, scraping of the walls of the tumor, and otoplastic operations. The latter can be performed with considerable freedom. The author's pet remedies, aconite in mild cases, and calcium sulphide "where suppurative processes arise" are, of course, not left without a good word.

The remainder of Chapter IX., twenty-two pages, gives a careful compilation of cases of gun-shot and sabre wounds of the ear, abstracted from "The Medical and Surgical History of Our Civil War," and from different American and foreign sources. They will be read with interest and prove useful for reference. In the author's own case (p. 169) an attempt at removal, under narcosis, of a bullet from the tympanum was unsuccessful. The patient was told to return, but failed to do so. Chapter X., "Injuries of the Drum-head and Drum of the Ear," is very instructive. Ruptures of the *Mt* from all possible causes, especially from explosions, are very fully discussed, and illustrated by drawings and the history of numerous cases. Chapters XI., "Anatomy," and XII., "Catarrhal Inflammation of the Middle Ear," are short, presenting nothing new.

Chapter XIII. is very readable, containing a number of suggestive remarks and some very instructive cases. It is strange, however, that the author continues denouncing the operations of opening the mastoid process. The almost universal approbation of this procedure has wrung from him the concession of its admissibility under certain conditions, but it seems that in all his 30,000 cases of ear disease he has never seen fit to perform it, his "success in affording drainage through the tympanum having deterred him as yet from the latter procedure" (p. 284). This procedure does not seem to us so strongly supported by his statement that fourteen of his cases of *acute* suppurative otitis have died (p. 274). In acute cases, where not only life but also good hearing can be preserved, where, through swelling of the soft parts in the drum cavity and ear canal, the secretion is stowed in the adjacent pneumatic cavities, seeking an outlet at the point of least resistance, which in a number of cases is the cranial cavity, it is easier, more efficient, more conservative, and safer to establish an outlet through the outer table of the mastoid process than

through the delicate and most important structures in and around the tympanic cavity. Moreover, the opening in the mastoid can readily be kept open until the inflammation has disappeared, whereas an incision through the soft parts surrounding the drum cavity is apt soon to close again.

The chapter on "Aural Polypi" is short and to the point. The Reviewer would only add that absolute alcohol, as recommended by Politzer, is useful not only after operations, but causes many young polypi to shrink and disappear; besides, it makes the operation of larger ones easier by lessening their bulk and contracting the swollen adjacent tissues. The Reviewer would further like to say that it is advisable, after each polypus operation, carefully to examine the base of the polypus, for recurrent polypi always spring from a carious or necrosed bone which ought to be scraped with a sharp spoon, and, if loose, removed with a pair of forceps.

Among the subsequent chapters, "Facial Paralysis," "Anomalies of Audition," "Aural Phenomena and Brain Affections," Chapter XVII., "Excision of the Drum-head and Ossicles," is the most important. It gives as well a conscientious appreciation of what, in this field, has been done by others (Schwartze, Kessel, Lucæ) as a detailed description of the author's views and experience, with a tabulated report of twenty-nine cases.

Part Fourth concludes the book with the following miscellaneous articles: The education of school children with defective hearing; the effect of high atmospheric pressure on the ear in tunnels, caissons, etc.; damage by unskilful attempts at removal of foreign bodies; claims of soldiers for pensions. H. KNAPP.

3. **A Manual of Diseases of the Ear.** By ALBERT H. BUCK. For the use of Students and Practitioners of Medicine. 419 pages. New York, W. Wood & Co., 1889. Price, $2.00.

Dr. Buck's manual may be considered a second, revised edition of his text-book, which, under the title "Diagnosis and Treatment of Ear Diseases," appeared in 1880 in "Wood's Library of Standard Medical Authors," where it was carefully withheld from the aural specialist. The present work, though bearing the original stamp of the author, is less personal than the former. The general descriptions are more systematic and concise, with a fair presentation of the views held by other, especially American, writers. The rules for diagnosis and treatment, as far as we can judge, are practical, trustworthy, and up to the times. We

think, however, that the author has clogged his text with the histories of too many cases from his practice. These histories may interest the specialist, but the "busy practitioner" and the busier student will read with attention only the general description of a disease and seek their personal experience rather in the clinics and their daily rounds than in the case-books of other physicians. Apart from that we may fairly say, that among the galaxy of text-books on the ear Dr. Buck's treatise holds an honorable position.

H. KNAPP.

MISCELLANEOUS NOTES.

On the occasion of the World's Fair this summer, an International Congress of Otology and Laryngology will be held in the Palais du Trocadéro, in Paris, from the 16th to the 21st of September. Committee of Organization: Prof. Duplay, President; Dr. Gougenheim and Ladreit de Lacharrière, Vice-Presidents; Dr. Löwenberg, Secretary.

The American Medical Association will meet this year at Newport, R. I., on the 25th of June, for a four days' session. The sections of ophthalmology and otology promise to be well attended.

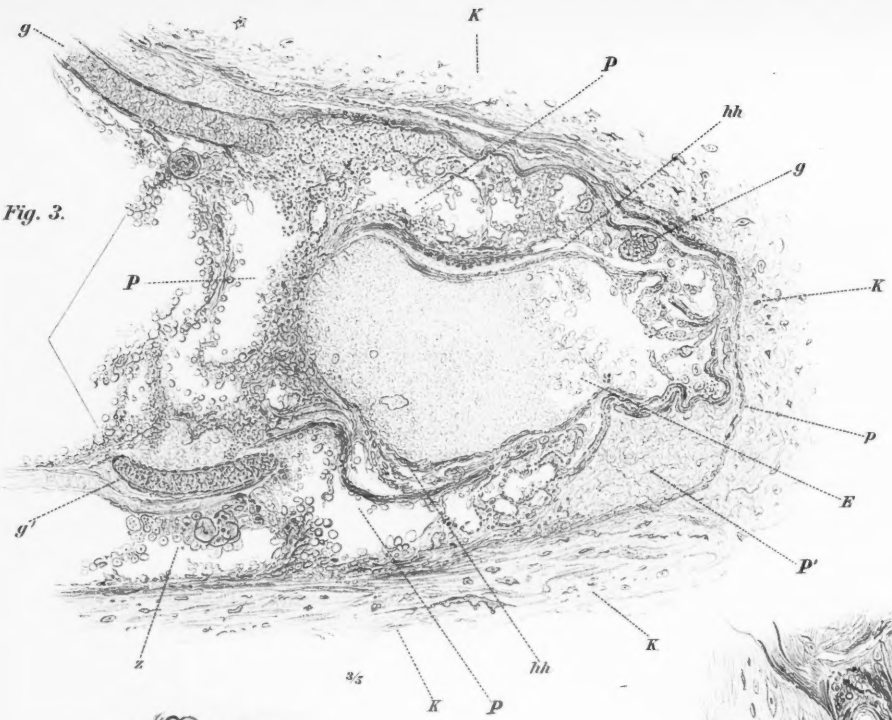


Fig. 3.

3/5

Fig. 4.

3/8

Fig. 5.

3/5

F. Veith, gez.

Fig. 2.

3/5

Fig. 1.

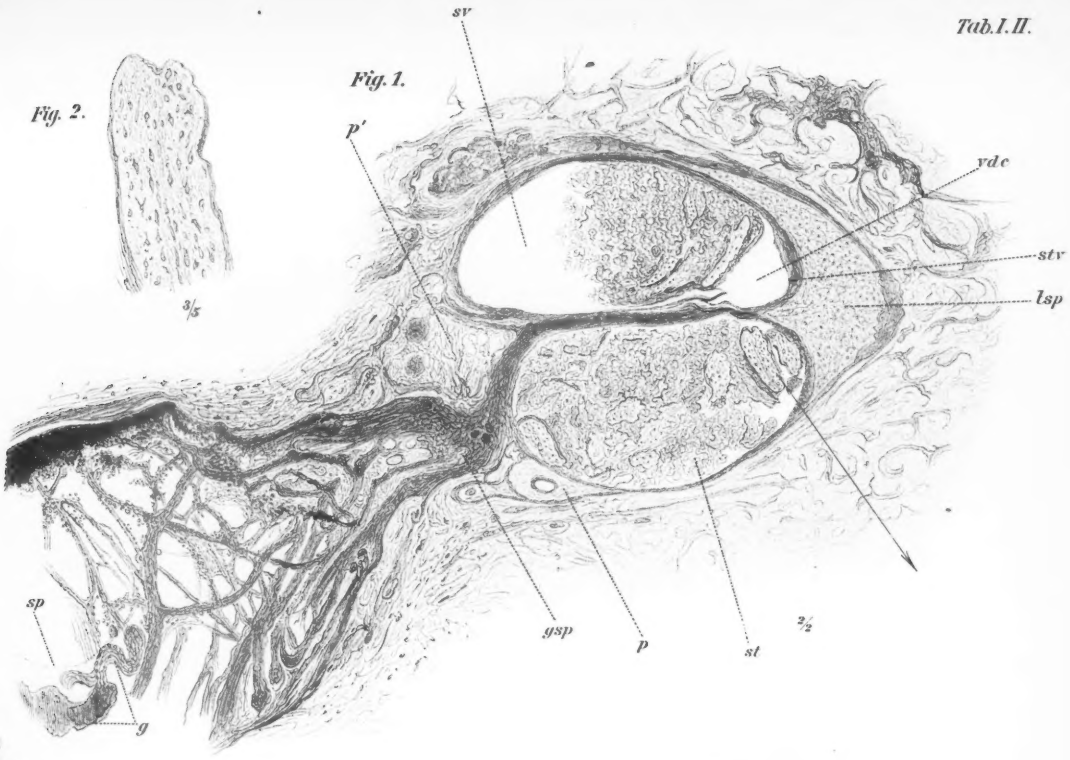
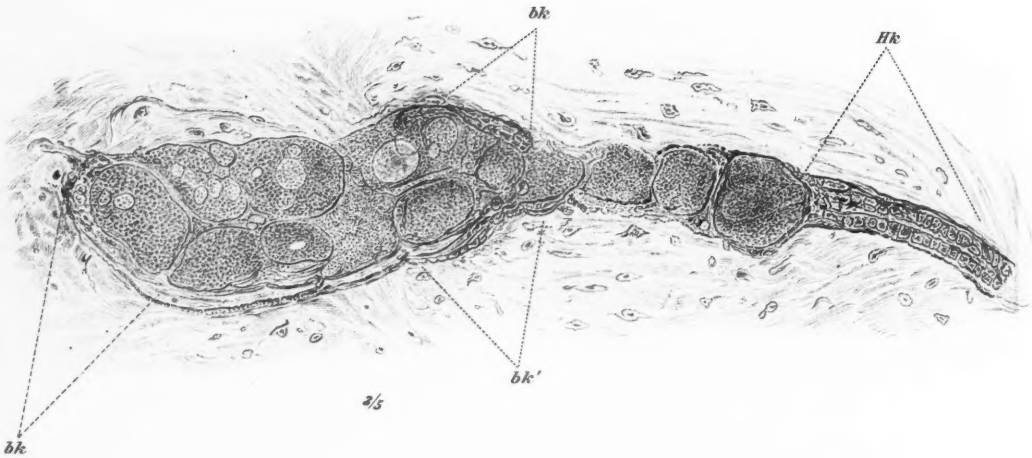


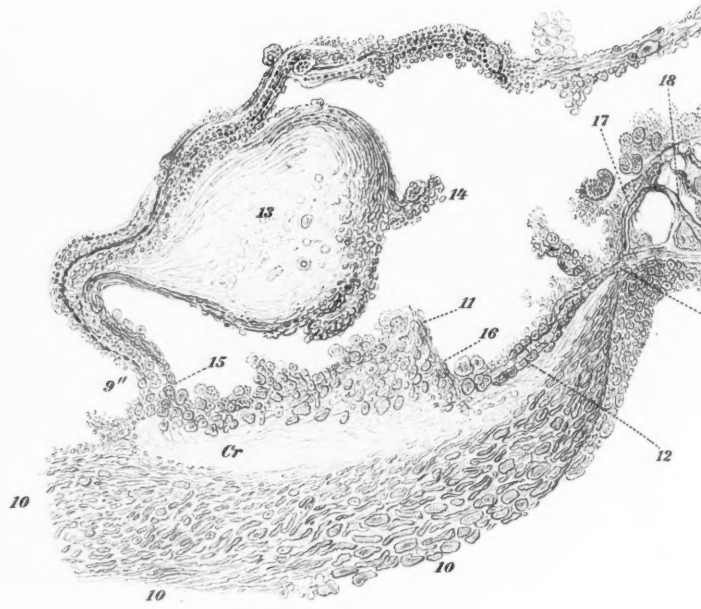
Fig. 6.

3/5

Lith. Anst. v. C. Kurst, Leipzig.

Fig. 7.





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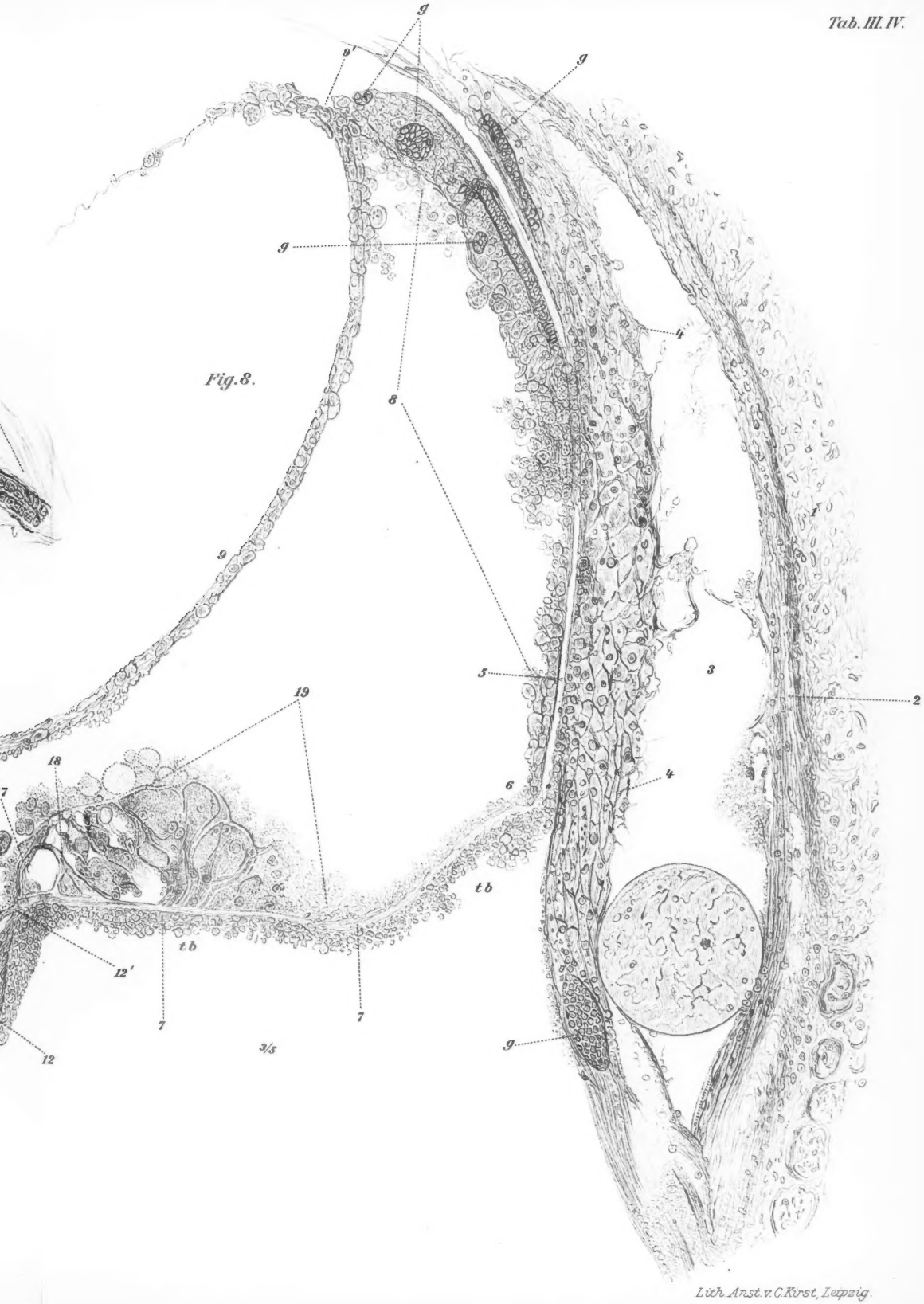

Fig. 8.

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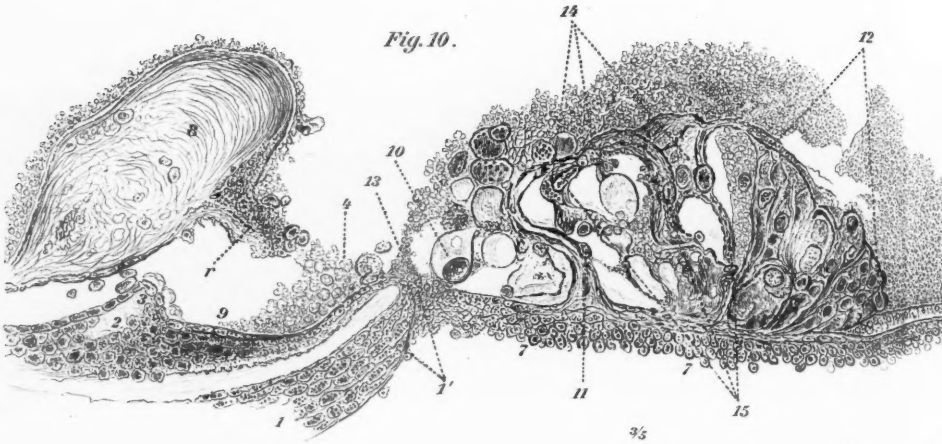
Fig. 10.



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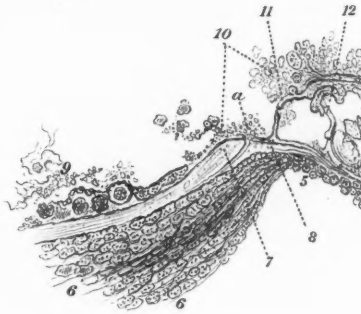

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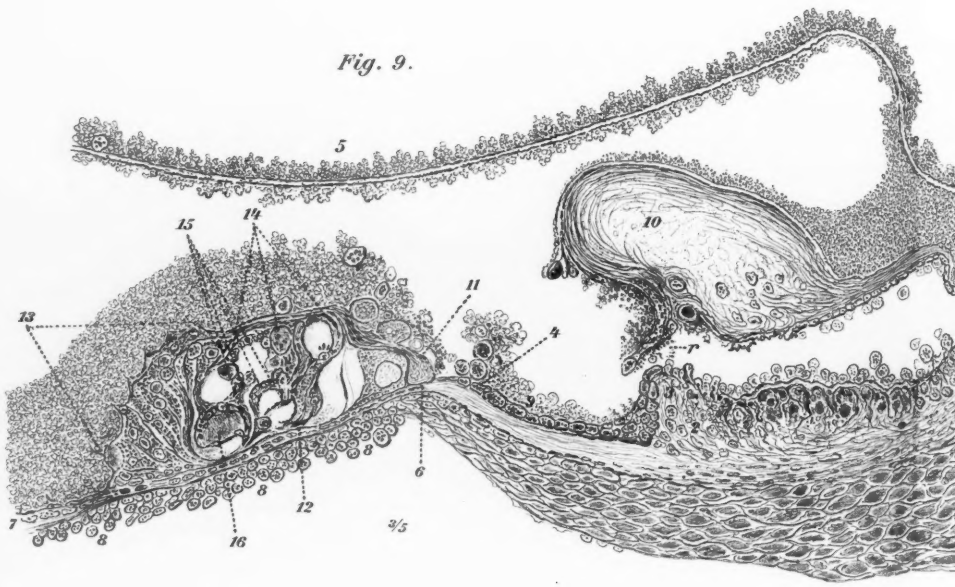

3/5

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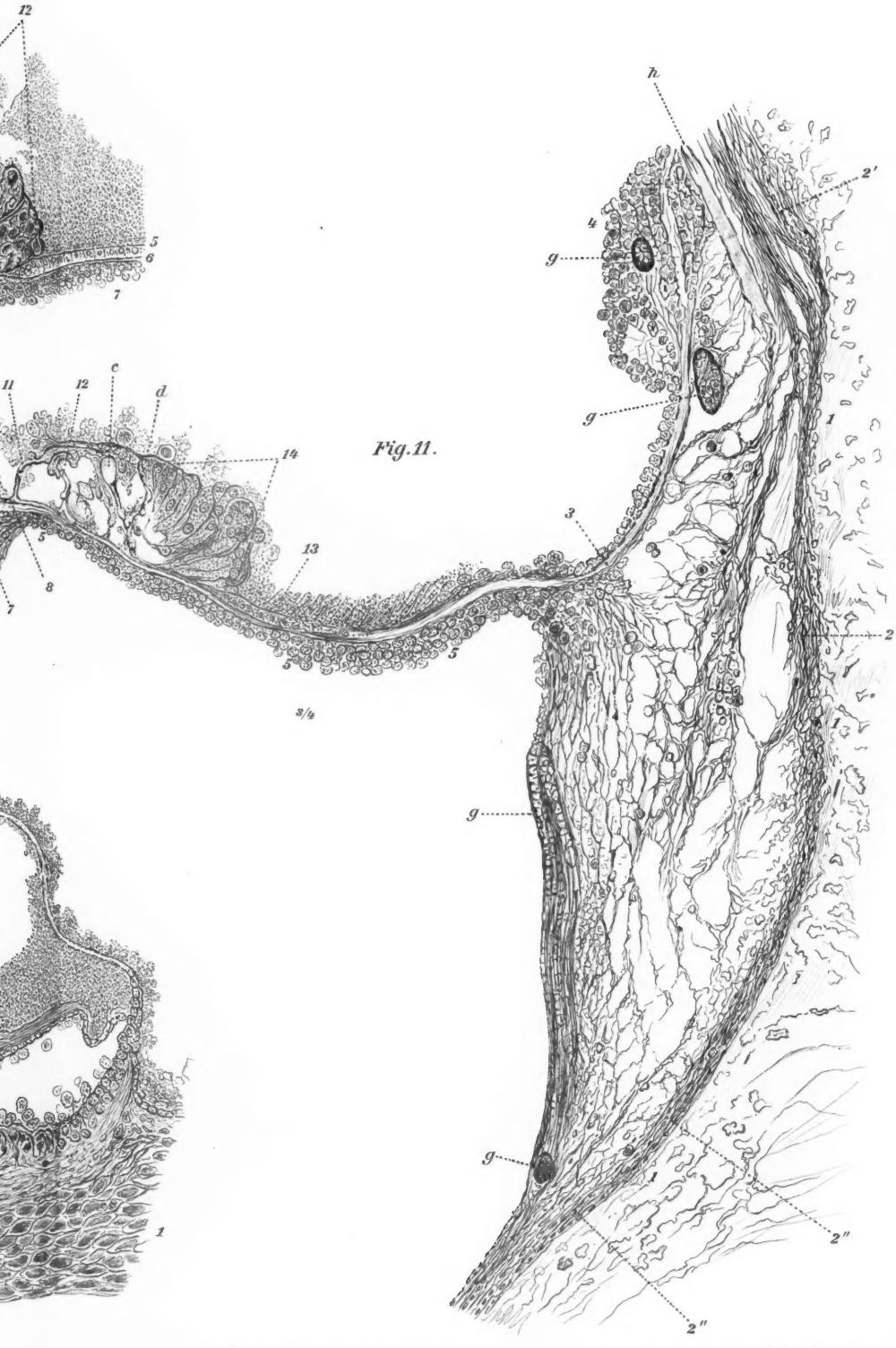

Fig. 11.

Lith. Anst. v. C. Kirst, Leipzig.

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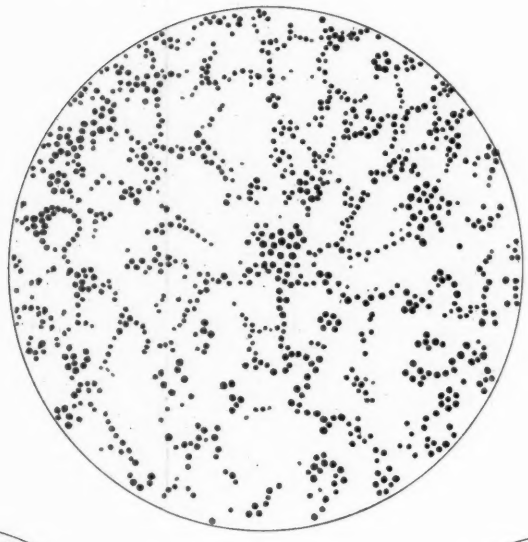
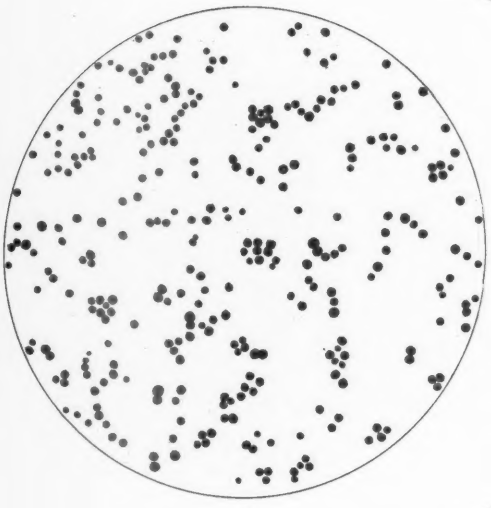


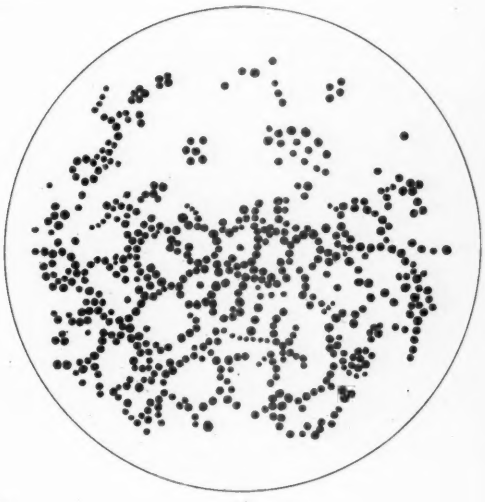
3/12

Fig. B.



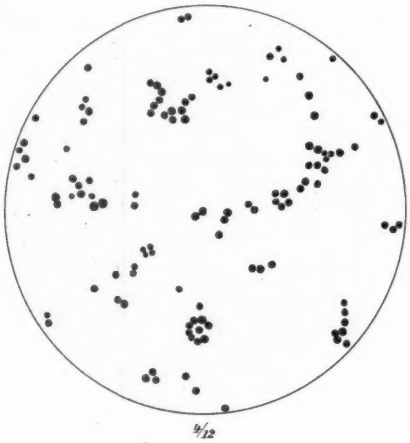
4/12

Fig. C.



4/12

Fig. D.



4/12

F. Voith. gez.

Lith. Anst. v. C. Kirst, Leipzig

CONTENTS OF VOLUME XVIII., NUMBER 2.

1. Case of Purulent Meningitis in which Purulent Disease of the Middle Ear Invaded the Interior of the Cranium through the Labyrinthine Cavities and Fallopian Canal. By Dr. Jas. Finlayson and Dr. Thos. Barr, of Glasgow 115

2. Necrosis and Caries of the Temporal Bone Treated with Acids. By Dr. Ole Bull, of Christiania, Norway 123

3. Report of Cases of Disease of the Mastoid Process, with Remarks. By Henry Ferrer, M.D., San Francisco, Cal. 139

4. Report on the Progress of Otology during the Second Half of the Year 1888. Translated by Dr. Max Toeplitz, New York . . 183

   I.—Normal and Pathological Anatomy, Histology, and Physiology of the Ear and Naso-Pharyngeal Cavity. By A. Barth, Berlin.

   A.—Anatomy.

   B.—Physiology and Physics.

   II.—Pathology and Therapeutics. By A. Hartmann, Berlin.

5. Miscellaneous Notes 215

   The Technique of Dissecting the Normal and Pathological Human Ear.

   Practical Guide to the Diagnosis and Treatment of the Diseases of the Nose. By Dr. G. Ferreri.

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